Chapter 11

Neurologic Disorders

John Pringle

I. **BRAIN DISORDERS OF THE NEWBORN.** There are many congenital defects of the nervous system of domestic animals. Most defects are lethal and are diagnosed at necropsy. Causes may include genetic or environmental factors. Environmental factors are highly varied and include teratogens, viruses, drugs, trace elements, and physical damage (e.g., rectal palpation of the dam).

A. **Hydranencephaly (normotensive** hydrocephalus) is an absence of cerebral hemispheres in a cranium of normal conformation.

1. Patient profile. This condition may be more common than hydrocephalus in large animals (see I B), particularly in calves because hydranencephaly is associated with intrauterine viral infection.

2. Clinical findings. Affected animals show signs immediately at birth, with depression and blindness (i.e., dummies) being the key findings. In the viral-associated hydranencephaly of calves, other problems such as cerebellar signs or arthrogryposis may predominate.

3. Etiology. In calves, the known causes include intrauterine viral infection by bovine viral diarrhea (BVD) virus, Akabane virus, or bluetongue virus. In some species, a fetal cerebrovascular accident has also been proposed as a cause, but this disease is otherwise poorly understood.

4. Diagnostic plan and laboratory tests. An accurate diagnosis in cattle, although challenging, is important for client education and prevention.
   a. Clinical signs of blindness and depression from birth are highly suggestive of hydranencephaly. Arthrogryposis in calves or lambs is suggestive of intrauterine viral infection as a cause.
   b. Presuckle serum titers that are positive to viruses, such as BVD virus in calves or bluetongue in calves or lambs, help confirm a diagnosis of intrauterine viral infection.

5. Therapeutic plan. Most affected animals lack vigor at birth and die or are euthanized.

6. Prevention. For viral hydranencephaly, vaccination of the dam before breeding can help prevent this problem. However, most cases are sporadic and are unlikely to occur at a high incidence.

B. Hydrocephalus (or hypertensive hydrocephalus) is the destruction of tissues within the cranial vault, usually caused by increased hydrostatic pressure in the cerebrospinal fluid (CSF).

1. Patient profile and history. Hydrocephalus can affect all animal species as an isolated occurrence, but it is also a rare inherited trait in cattle. Hydrocephalus has been associated with dwarfism in cattle.

2. Clinical findings. The animal may be born dead. If the affected animal lives, it is blind and very depressed from birth and usually dies within a few days. Other possible signs include a "domed" cranial enlargement, microphthalmia, and reduced birthweight.

3. Etiology and pathogenesis
   a. Etiology. This disease is caused by a simple autosomal-recessive trait in cattle (particularly Herefords) or a vitamin A deficiency in cattle.

b. **Pathogenesis.** Hydrocephalus occurs when there is an accumulation of excessive fluid within the ventricular system.

4. **Diagnostic plan**
   a. Clinical signs of a domed skull, depression, and blindness from birth suggest hydrocephalus.
   b. Ultrasound examination of the cranial vault through fontanelles in the skull confirms a lack of brain tissue.

5. **Therapeutic plan and prevention.** Although there is no treatment for the affected animal, genetic planning should be considered if cases are found in purebred Hereford calves.

C. **Cerebellar disease.** Congenital cerebellar diseases can be classified as neonatal syndromes, which are present at birth, or postnatal syndromes, which may develop weeks to months after birth. Neonatal syndromes are most common.

1. **Patient profile.** All species can be affected. Postnatal syndromes have been recognized in Arabian foals, Gotland ponies, Holstein cattle, and Yorkshire pigs.

2. **Clinical findings** consist primarily of a lack of control of voluntary movement. The neonatal syndromes occur during fetal development and have no progression of signs after birth. Compensation for the deficit may occur over several weeks.
   a. A key finding in pure cerebellar disease is strength without control, with no paresis or proprioceptive disturbances. Intention head tremors are often present and are particularly obvious when the animal moves its head to eat.
   b. Ataxia, **hypermetria** (or hypometria), and **spasticity** are the major features of gait.
   c. A unilateral or bilateral lack of menace response may be present, despite normal vision and normal cranial nerve VII function, because cerebellar processing is required in the normal menace response.

3. **Etiology and pathogenesis**
   a. Neonatal syndromes
      (1) **Infectious** causes are the most common cause of neonatal syndromes.
         (a) **Cattle.** BVD virus in utero infection at 100–170 days' gestation or intrauterine bluetongue virus infection are known to cause cerebellar hypoplasia in cattle.
         (b) **Sheep.** Border disease virus intrauterine infection at 60–80 days' gestation, as well as bluetongue virus, can result in cerebellar disease in sheep.
         (c) **Pigs.** Hog cholera virus infection in pigs also causes cerebellar damage.
      (2) Malformation can also lead to neonatal cerebellar disease and has been associated with an autosomal-recessive gene in Hereford cattle.
   b. Postnatal syndromes are classified as abiotrophies because they are characterized by lesions of degeneration. Abiotrophies are genetically-induced defects in cerebellar cortical neurons that result in the premature death of these neurons.

4. **Diagnostic plan and laboratory tests.** Clinical signs of intention tremors, hypermetria, and spasticity without paresis or decreased sensorium are usually sufficient to suggest cerebellar disease. The time of occurrence (at birth or postnatally) provides further direction regarding cause (e.g., intrauterine or postnatal abiotrophy).
   a. Neonatal syndromes
      (1) **Serology.** In cases of cerebellar disease at birth, precolostral serology (e.g., BVD in calves) helps establish a viral etiology.
      (2) **Postmortem** examination
         (a) Intrauterine viral infection. The cerebellum is absent, reduced, or normal in size, with histologic evidence of Purkinje cell loss.
         (b) **Malformation.** The cerebellum is significantly smaller than normal.
   b. Postnatal syndromes. The cerebellum of the affected animal is of normal size.

5. **Therapeutic plan.** There is no treatment for affected animals, but the clinical abnormalities do not usually limit animal survival or growth.

6. **Prevention.** If a virus is incriminated, the vaccination of dams can prevent future disease.

D. **Neonatal maladjustment syndrome [NMS; "Barker foals," hypoxic–ischemic encephalopathy (HIE)]**

1. **Patient profile.** This syndrome occurs in foals, most commonly in Thoroughbreds. Foals may be premature or may be from a difficult or prolonged delivery.

2. **Clinical findings.** After an initial period of apparent normality (approximately 24 hours), affected foals lose their affinity for the dam, lose suckling ability, and may wander aimlessly. They may also experience seizures with opisthotonos, appear unaware of their surroundings, and lose their righting reflex.

3. **Etiology and pathogenesis.** The etiology of NMS has not been fully determined. It is felt that hypoxia at parturition or trauma to the central nervous system (CNS) at birth with resulting vascular abnormalities and cerebral hemorrhage may be part of the pathogenesis (see Chapter 18 III A 2). There appears to be poor correlation of clinical signs with severity of brain hemorrhage.

4. **Diagnostic plan and laboratory tests**
   a. **Patient history.** The clinical history of loss of suckle on the first day after initial appearance of a normal foal is suggestive of NMS.
   b. **Laboratory tests.** There are currently no diagnostic tests to detect brain hemorrhage. However, tests for failure of passive transfer using zinc sulfate turbidity or a commercially available test (e.g., Cite Test), as well as a complete blood cell count (CBC) and CSF analysis should be performed to exclude sepsis or meningitis. A blood glucose level should rule out hypoglycemia.

5. **Differential diagnoses.** Other causes of loss of suckle can include sepsis and metabolic disturbances. Seizures can occur with hypoglycemia or meningitis.

6. **Therapeutic plan**
   a. Because sepsis or hypoglycemia can occur concomitantly with NMS, any suggestion of these disorders on laboratory samples is sufficient evidence for treatment with plasma, antibiotics, and/or intravenous glucose.
   b. NMS. Seizures should be controlled using **diazepam** or **phenobarbital. Dexa**methasone or dimethylsulfoxide (DMSO) can be used in animals with suspected cerebral hypoxia caused by brain hemorrhage. DMSO may be the drug of choice if sepsis is also a possibility.
   c. Supportive care. The foal should receive supportive care, including tube feeding, deep soft bedding, and possibly antibiotics and plasma transfusion in the case of failure of passive transfer.

7. **Prognosis.** The prognosis for recovery is guarded if other diseases, such as sepsis, are present. Otherwise, there is a good prognosis (more than 80% recovery rate) with supportive care.

E. Bacterial meningitis (meningoencephalitis)

1. **Patient profile.** Bacterial meningitis is seen in neonates of all species.

2. **Clinical findings** vary depending on the stage of infection.
   a. Initial clinical signs include depression and fever. These rapidly progress to signs of hyperirritability (e.g., hyperesthesia, hyperalgesia, opisthotonos, convulsions) and eventually coma.
   b. Other signs include muscular rigidity of neck, diarrhea, joint ill (i.e., arthritis), omphalophlebitis, uveitis, or hypopyon.

3. **Etiology and pathogenesis**
   a. **Etiology.** Meningitis in neonates is most often secondary to septicemia. Septicemic strains of Escherichia coli are the most common cause, with the exception of Streptococcus suis type II, which is the most common cause in piglets.
   b. **Predisposing factors.** Failure of passive transfer of immunoglobulins increases

an animal's susceptibility to septicemia and meningitis. In addition to a lack of colostral transfer, predisposing factors include enteritis, omphalitis, or respiratory infections.

c. Pathogenesis. Portals of entry for organisms include the pharynx, intestinal tract, and navel. The route of entry to the meninges (leptomeninges) is then hematogenous. Some organisms (e.g., *S. suis* type II in piglets) can infect animals via the cribriform plate.

4. Diagnostic plan
   a. CSF analysis. A CSF sample (either atlantooccipital or lumbosacral) will show a moderate to high protein content and elevated white blood cell (WBC) count in animals with bacterial meningitis. Culture of the CSF is not always positive but should be attempted.
   b. Blood work. A CBC often reveals a neutrophilic leukocytosis with or without left shift, but this is not diagnostic for meningitis because other organs may also be involved in the septic process.
   c. Clinical chemistry. A sodium sulfite turbidity test or single radial immunodiffusion (SRID) kit (for calves) often identifies hypogammaglobulinemia, which results from the failure of passive transfer.

5. Therapeutic plan. When clinical signs are clearly present, therapy is often unrewarding, and the mortality rate is high, despite appropriate treatment.
   a. Early treatment with broad-spectrum antibiotics (e.g., synthetic penicillins, aminoglycosides) can be attempted.
   b. Pain relief with aspirin (25 mg/kg every 12 hours) or flunixin meglumine (2.2 mg/kg intravenously every 12 hours) should be given.
   c. Supportive care includes soft, dry bedding and the administration of nutrition and intravenous fluids during the treatment period.

6. Prevention. The key to prevention of meningitis in neonates is attention to parturition to ensure adequate colostral intake.

II. BRAIN DISORDERS OF MATURE AND GROWING ANIMALS

A. Congenital disorders, such as cerebellar disease (see I C) and Arab foal idiopathic seizures (see II C 4) can manifest in older animals.

B. Traumatic disorders
   1. Patient profile and history. There is often a history of head trauma, which is particularly common in young foals that flip over backwards when they are learning to be led.
   2. Clinical findings
      a. Main clinical signs include depression or coma.
      b. Accompanying signs may include CNS deficits, such as anisocoria (inequality of pupillary diameter), head tilt, nystagmus, and selected cranial nerve deficits, depending on the site of trauma. If the animal is ambulatory, ataxia may also be present.
   3. Pathogenesis Cranial trauma causes membrane disruption and cellular swelling that proceeds to cerebral edema, with increased intracranial pressure and tissue hypoxia. If a basal fracture occurs, then intracerebral hematoma formation may occur, resulting in focal neurologic signs. These neurologic signs vary depending on the site of the hematoma. However, a localized hematoma may be hard to distinguish because of the more generalized brain disease caused by the intracranial edema.
   4. Diagnostic plan. A key aspect of diagnosis of head trauma is usually the direct observation of the event or the sudden onset of clinical signs in an otherwise normal animal.

   a. Skull radiographs should be taken to check for fractures, but these may not be diagnostic if the fracture occurs along the epiphyseal lines and is not displaced.
   b. CSF analysis may reveal acute frank hemorrhage into the normally clear colorless fluid. If the trauma is less acute, there may be yellow discoloration to the CSF sample (xanthochromia), which is suggestive of blood breakdown products in the fluid. A CSF sample should not be taken if increased CSF pressure is present because doing so could result in further brain damage.

5. Therapeutic plan
   a. A patent airway must be established in comatose patients.
   b. Cerebral edema reduction
      (1) Drugs that may reduce cerebral edema include dexamethasone (1–2 mg/kg intravenously every 6 hours) or DMSO (0.9–1.0 g/kg of 10% solution in 5% dextrose intravenously).
      (2) Mannitol (0.25–2 g/kg of 20% solution intravenously), an osmotic diuretic, is also advocated but could cause increased swelling if there is significant vascular leakage in the brain tissue, allowing the mannitol to escape the brain vascular space.
   c. Seizure control. If convulsions occur, diazepam (25 mg for foals, 100 mg for adults intravenously) reduces the signs of seizures and helps prevent further self-induced trauma.
   d. Antibiotics should be administered if there are open fractures on the head.

6. Prognosis. With severe head trauma, the prognosis for the return of full function is grave.

C. Disorders characterized by seizures. Seizure disorders may occur in all large animals for a variety of reasons. Causes may be idiopathic, metabolic, traumatic, infectious, nutritional, degenerative, neoplastic, or inflammatory. Treatment is directed at resolving the cause of the seizure. In the case of idiopathic seizures, seizure control is the primary focus of treatment.

   1. Polioencephalomalacia (PEM, cerebral cortical necrosis)
      a. Patient profile and history
         (1) Patient profile. This disease affects cattle, sheep, and goats. PEM can affect an individual or appear as a herd problem, affecting calves ages 6–12 months and lambs and kids ages 2–6 months. PEM is usually seen in cattle in spring or early summer under feedlot conditions. PEM is less common in adults, and it is more sporadic in small ruminants.
         (2) History. Affected animals often have a history of some feed change or access to increased carbohydrates in the feed.
      b. Clinical findings are referable to cerebral edema and laminar necrosis.
         (1) Cortical blindness (i.e., absent menace with intact palpebral and pupillary light reflex) occurs early in the course of disease.
         (2) Dorsomedial strabismus and nystagmus are also common findings with PEM.
         (3) Other neurologic signs include incoordination, muscle tremors, and depression, which is interspersed with hyperexcitability leading to recumbency, opisthotonos, and paddling with extensor rigidity.
         (4) Vital signs can be normal or elevated because of exertion. The rumen usually remains active.
      c. Etiology and pathogenesis
         (1) Etiology. PEM is the result of thiamine deficiency. Thiamine deficiency can result from:
            (a) Decreased thiamine synthesis by rumen microorganisms
            (b) Increased thiamine destruction by thiaminase (found in bracken fern, horsetail)
            (c) Rumen microbial destruction of thiamine by Bacillus *thiaminolyticus* or *Clostridium sporogenes*
            (d) Thiamine antimetabolites (i.e., amprolium)
         (2) Predisposing factors. Feeding high concentrate, low-roughage diets has

been linked to PEM, as has high-sulfate dietary or water sources. Major management changes in feeding (particularly with high concentrate diets) may favor the development of thiaminase-producing bacteria in the rumen.

(3) Thiamine is necessary for the production of red blood cell (RBC) transketolase, a major enzyme in the metabolism of glucose via the pentose phosphate pathway. It is postulated that ruminants have increased requirements for glucose via this pathway at the level of the brain.

d. Diagnostic plan and laboratory tests
(1) Response to treatment. Apart from the clinical findings, particularly cortical blindness and a history of feed change or high-carbohydrate feeding, a key method of diagnosis is the response to specific medical treatment (e.g., thiamine).
(2) Assays for metabolic **abnormalities.** Most routine laboratory findings are not diagnostic, and only in specialized laboratories is it possible to assay for the metabolic abnormalities. These abnormalities include low blood thiamine and decreased erythrocyte transketolase.
(3) CSF analysis. The protein level can be normal to highly elevated, and the cellularity varies from slight to severe mononuclear pleocytosis. The CSF pressure is increased to 200–350 mm of saline (normal pressure is 120–160 mm saline). However, none of these CSF changes are diagnostic for PEM.
(4) Necropsy
(a) If an animal dies, a key finding for a presumptive diagnosis is **autofluorescence** of a freshly cut surface of brain cortex when placed under ultraviolet light.
(b) At necropsy, there is diffuse cerebral edema with compression, yellow discoloration of the dorsal cortical gyri, and laminar necrosis of cerebrocortical grey matter.

e. Differential diagnoses. Diseases that should be ruled out include lead toxicity, pregnancy toxemia in sheep, and nervous ketosis in dairy cattle.

f. Therapeutic plan
(1) Thiamine (10 mg/kg intravenously early in the course of clinical signs), followed by intramuscular or subcutaneous dosing every 6 hours is the drug of choice. A positive response (i.e., the reduced severity of neurologic signs and improved mentation) is seen within 24 hours if the thiamine is administered early in the course of disease.
(2) **Corticosteroids,** mannitol, or DMSO may be indicated for animals with possible cerebral edema.
(3) **Tranquilizers.** Affected animals that suffer convulsions should be tranquilized to prevent self-induced trauma.
(4) Supportive care. Animals should receive supportive care, including soft, dry bedding while recumbent and parenteral or oral fluid administration.
(5) Diet When the affected animal shows an interest in eating, it should be given only roughage for several days before being reintroduced to concentrates.
(6) Euthanasia. If no response to treatment is seen within 3 days, euthanasia is warranted because there is likely permanent brain damage.

g. Prevention. Although PEM occurs sporadically, the risk of the disease can be reduced by avoiding sudden feed changes.

2. **Lead** poisoning
a. Patient profile. Cattle, sheep, and, less commonly, horses can be affected by lead poisoning, with problems more likely to occur in young animals.
b. Clinical findings, Three forms, which are somewhat dose dependent, are described.
(1) Acute form
(a) The acute form most commonly affects young cattle. Affected animals show a sudden onset and short duration of disease (12–24 hours).
(b) Clinical signs include staggering, muscle tremors, clamping of the jaws, frothing at the mouth, snapping eyelids, rolling eyes, cortical blindness,

---

head pressing, aggressive behavior, and convulsions. Sudden death can also be a finding of the acute phase.
(2) Subacute form
(a) The subacute form is found in both cattle and sheep.
(b) Clinical signs include dullness, anorexia, blindness, circling, abdominal pain, muscle tremor, and constipation followed by diarrhea. Signs of gastroenteritis include ruminal atony (accompanied by constipation in early stages), followed by fetid diarrhea due to abomasitis from lead salts.
(3) Chronic form
(a) The chronic form is reported in horses.
(b) Clinical signs. A degeneration of peripheral nerves usually manifests as paralysis of the recurrent laryngeal nerve and the pharynx. This paralysis may result in recurrent choke, regurgitation of food, and aspiration pneumonia.

c. Etiology and pathogenesis
(1) Etiology. Lead has been one of the most common forms of toxicity in farm animals.
(a) Cattle with indiscriminant appetites often ingest toxic quantities in short periods of time. Sources of lead include lead-bearing paints, car batteries, lead shot, and solder or leaded windows.
(b) The reported source of chronic lead toxicity is the grazing of pastures near highways that have leaded gasoline contamination; however, with the production of all lead-free gasoline, this disorder may soon become less common in North America. Environmental pollution from smelters has also been incriminated.
(2) Pathogenesis. After lead is ingested and absorbed, it localizes in capillary endothelial cells. The major lesion is vascular, leading to cerebral congestion, edema, and cortical necrosis. Lead also interferes with cell function.

d. Diagnostic plan and laboratory tests
(1) Clinical signs, including the jaw champing and snapping closed of the eyelids, are highly suggestive of lead toxicity.
(2) Blood work. A CBC may reveal nucleated erythrocytes, but the classic basophilic stippling reported in other species does not occur very frequently in large animals. Whole blood lead levels of 0.35 ppm or greater are considered indicative of lead toxicity in cattle.
(3) Postmortem examination. Finding lead in renal tissue can confirm the diagnosis.

e. Differential diagnoses. Other neurologic diseases that should be considered include hypovitaminosis A, hypomagnesemic tetany, brain abscesses, poisoning due to arsenic, mercury, or *Claviceps paspali* (ergotism), listeriosis, polioencephalomalacia, and thromboembolic meningoencephalitis. A key clinical sign that helps differentiate lead toxicity from polioencephalomalacia is the finding of an atonic rumen—rumen motility should be normal in polioencephalomalacia.

f. Therapeutic plan
(1) Removal from access to lead sources may be the only change necessary to halt an outbreak because the slow reversal of signs can occur.
(2) Therapeutic agents
(a) Clinically affected individual animals can be treated with calcium **disodium** ethylene diamine tetra-acetate (EDTA) administered by slow intravenous injection as a 6.6% solution (1 ml109 kg daily in two or three divided doses). This drug chelates osseous lead and hastens urinary excretion.
(b) Magnesium sulfate given orally may aid in precipitating soluble lead salts that remain in the intestine.
(c) Thiamine is also thought to help by decreasing lead deposition in soft tissue.
(3) Nursing care is important, particularly to control seizures that may result in self-induced trauma.

g. Prevention. Most cases of lead poisoning result from the careless disposal of

lead-containing materials. Proper disposal of these toxic materials aids in prevention.

h. Economic **implications.** Carcasses that contain high lead levels may be rejected at slaughter. After lead toxicity in cattle, 6 months may be required for tissue lead levels to drop to background levels.

3. Salt poisoning (water deprivation)

a. Patient profile. Salt poisoning can occur in all species but has been best studied in swine. Because the main cause is a history of water deprivation, combined with high dietary salt intake (e.g., sodium chloride in the feed or the sole water source), salt poisoning is more likely to occur in confined animals.

b. **Clinical** findings

(1) Affected animals are depressed, may appear blind, wander aimlessly, and exhibit head pressing. They are unresponsive to stimuli and may pivot around a front or hind limb.

(2) Intermittent convulsive seizures may occur, becoming increasingly frequent until the animal becomes comatose and dies.

(3) Characteristic signs in pigs include a loss of squeal and "walking-backward" fits.

c. Etiology and pathogenesis

(1) Etiology. Water intake can be reduced because of mechanical problems in automatic waterers, frozen water, overcrowding, or refusal to consume medicated water. Drought (resulting in restricted access to fresh water) and high salt intake from water sources contaminated with salt (such as in regions with oil fields) have been associated with this disease in cattle at pasture.

(2) Pathogenesis

(a) Increased body sodium levels cause a metabolic blockade and inhibit anaerobic glycolysis at the cellular level, resulting in decreased energy production in the brain and associated depression.

(b) To compensate for this high sodium level, brain cells begin to produce osmotic products called "idiogenic **osmoles,**" which, over time, change the osmotic gradient to reduce the deleterious influx of the excess sodium ion into brain cells. When water is given to the affected animal in an effort to reduce serum sodium levels (by hemodilution), the osmotic gradient maintained by these idiogenic osmoles causes cell swelling and brain edema, leading to edema, increased intracranial pressure, and subsequent malacia. Seizures and coma are resulting signs.

d. Diagnostic plan and laboratory tests

(1) Serum and CSF sodium levels above 160 mEq/L are suggestive of salt poisoning and water deprivation, with higher levels (170 mEq/L and over) sufficient for a presumptive diagnosis.

(2) Histopathology. An eosinophilic meningoencephalitis is found on histopathology only in swine; otherwise, microscopic changes are usually nondiagnostic.

e. **Therapeutic** plan. When the disease is recognized, the animal should be given small amounts of fresh water per os at frequent intervals. Excessively rapid reduction of sodium levels can result in severe seizures and brain edema. Recovery, if it occurs, may take 4–5 days.

f. Prognosis. The prognosis for recovery when blood sodium levels are markedly elevated is grave for all affected species.

g. **Prevention.** Ensuring unlimited access to fresh water prevents most cases of salt toxicity. For calves, proper dilution of oral electrolyte solutions also helps avoid this disorder.

4. Arab foal idiopathic seizure

a. Patient **profile.** This uncommon disease occurs in Arab foals with predominantly Egyptian bloodlines.

b. Clinical findings. Frequent seizures are preceded by prodromal signs. The seizures may be initiated during attempts to train or discipline foals. Between seizures, affected foals are clinically normal.

c. Etiology. The cause of Arab foal idiopathic seizure is unknown but, because this disease is seen in a particular bloodline, genetics may play a role. This disease should be considered as a differential diagnosis in Arabian foals that have neurologic signs that may be confused with environmentally-induced problems.

d. Diagnostic plan and laboratory tests. All laboratory data (e.g., blood, CSF analysis) are within normal limits. A diagnosis is made based on the exclusion of other seizure causes (e.g., trauma, metabolic disturbance) and the appropriate bloodlines of the foal.

e. Therapeutic plan. The only treatment available is symptomatic. Diazepam is used to control seizures during the clinical episodes. Most animals appear to outgrow the seizures by ages 6–8 months.

D. Disorders **characterized** by behavioral and personality changes

1. Thromboembolic meningoencephalitis (TEME)

a. Patient profile. This disease occurs in feedlot cattle of any age but is most common in animals ages 4–12 months, particularly in fall and winter. Signs may begin to appear approximately 4 weeks after arrival to feedlot with several animals affected. The morbidity rates in a herd are low (2%), whereas case **fatality** rates are high (90%).

b. Clinical findings

(1) **Peracute** form. Cattle may be found dead.

(2) Acute form. Early signs include separation from other cattle with depression and apparent blindness. The rectal temperature can be very high, at 41°C–42°C. Anorexia, staggering, recumbency and coma progressing to lateral recumbency, opisthotonos, and partial paralysis of multiple cranial nerves may be present.

(a) Less severe signs can include polyarthritis, respiratory disease, and occasionally otitis media in affected animals or in penmates.

(b) Ophthalmologic examination shows retinal hemorrhages (highly suggestive of TEME) in approximately 20% of affected animals.

c. Etiology and pathogenesis

(1) Etiology. The cause of TEME is bacteremia due to *Haemophilus somnus.* H. somnus infections can occur in a respiratory, urogenital, or septicemic form. The neurologic signs associated with the TEME are part of the septicemic form of disease.

(2) Pathogenesis

(a) **Transmission** is thought to be via respiratory or urogenital secretions from presumed carrier cattle.

(b) The pathogenesis of TEME involves a **severe vasculitis.** rather than an embolic phenomenon. It is not known whether the suppurative vasculitis is caused by bacterial attachment to the endothelial cell, leading to destruction of the cell, or whether endotoxin and exotoxin release are responsible for the vasculitis. Animals with TEME may have lesions in the heart, liver, kidney, and joints.

d. Diagnostic plan. The clinical findings of sudden onset of neurologic signs with high fever, accompanied by respiratory and joint involvement in feedlot cattle, are highly suggestive of this disease.

(1) CSF analysis. A CSF sample reveals marked pleocytosis with neutrophils predominating, along with high protein levels (i.e., a positive Pandy test).

(2) Culture of H. *somnus* from CSF, blood, synovial fluid, urine, or a tracheal wash can be attempted, but because of specific growth requirements, this test may be unrewarding.

(3) Serologic testing for infection can be equally frustrating to interpret because of seroconversion in asymptomatic animals.

(4) Postmortem examination

(a) Histopathology of the brain shows a severe vasculitis with neutrophilic infiltrates and occasionally vascular thrombi. Hemorrhaging retinal vessels, which reveal the vasculitis, are sometimes considered to be patho-

gnomonic for **TEME.** Unfortunately, because many affected animals lack these changes, this finding is an insensitive indicator of the disease.

- **(b)** Multifocal areas of hemorrhagic necrosis are found in the brain and spinal cord.
- **(c)** There may be a fibrinopurulent meningitis, polyarthritis, laryngitis, tracheitis, retinitis, conjunctivitis, and endometritis.

e. Differential diagnoses include polioencephalomalacia, lead toxicity, and salt poisoning/water intoxication.

f. Therapeutic plan. If affected animals are treated before they become recumbent, they can recover within 6–12 hours. The drug of choice is **oxytetracycline (20 mg/kg** intravenously every 24 hours for three days). However, the causative organism is not a highly resistant species and is likely to respond to other antibiotics if chosen.

g. Prevention. A killed bacterin has been used as a preventative with some success for herd protection. However, given the low morbidity rates associated with this disease, vaccination may not be economically feasible.

2. **Nervous** coccidiosis

a. Patient profile. This disease is seen in calves and young cattle, particularly in feedlots. Nervous coccidiosis is almost exclusively a winter disease and occurs mainly in the northwestern United States and western Canada.

b. Clinical findings
- (1) The key neurologic findings are ataxia, muscle tremor, blindness, and hyperexcitability accompanying intermittent or continuous seizures. Seizures may be precipitated by strenuous handling. Calves become recumbent with signs of opisthotonos, tonic–clonic movement, medial strabismus, and snapping of the eyelids.
- **(2)** Affected animals also show blood flecks in feces or blood-tinged diarrhea, which is associated with intestinal coccidiosis.

c. Etiology and pathogenesis. A heat-labile neurotoxin has been identified in the serum of affected calves, but not in the serum of calves with only intestinal coccidiosis. The nature of the toxin is unknown, and the associated mechanism linking nervous coccidiosis to intestinal coccidiosis has not been determined.

d. Diagnostic plan and laboratory tests. The main method of diagnosis is to **rule** out other possible diseases (see II D 5). A presumptive diagnosis can be made if the location and climate are suggestive of nervous coccidiosis (i.e., cold winter months in western Canada or the northwestern United States).
- **(1)** Microscopic examination of feces is used to confirm enteric coccidiosis but is not specific for the nervous form.
- **(2)** **Hyperglycemia,** hypochloremia, and low liver copper and iron stores have been found in affected animals.

e. **Differential** diagnoses include lead toxicity, PEM, TEME, or salt and water intoxication, for which there are more sensitive and specific diagnostic tests.

f. Therapeutic plan
- (1) Sulfonamides and supportive fluid therapy can be administered to treat intestinal coccidiosis.
- **(2)** Tranquilizers. Animals with convulsions can be treated by sedation to Prevent self-induced trauma.
- (3) Nursing care, including deep bedding and dry shelter, is important.
- **(4) Iron** or copper salt therapy. It is not known whether iron or copper salt therapy is of any benefit.

g. **Prognosis.** Affected calves have a poor prognosis for survival, with a 70%–100% mortality rate expected and death often occurring within 5 days of disease onset Undoubtedly, part of the mortality may be attributed to the low level of nursing care possible in these management conditions because of the harshness of the climate when the disease is observed.

h. **Prevention.** Because little is known about the risk factors involved, there are no known preventative measures.

3. Listeriosis

a. Patient profile and history
- (1) Patient profile. This disease occurs in all ruminants but is extremely rare in horses. Listeriosis generally affects animals younger than 3 years and is sporadic in cattle, with sheep demonstrating a higher morbidity.
- (2) History. Heavy silage feeding may be a feature of the history, but disease can also occur in animals at pasture.

b. Clinical findings
- (1) The neurologic signs can occur gradually or suddenly, with dullness, head tilt, and circling (hence the name "circling disease" in cattle) as common findings.
- (2) Other signs can include lateral inclination of head and unilateral facial nerve paralysis, dropped jaw, inability to swallow, and pyrexia. Less commonly, Listeria species cause septicemia in monogastrics or abortion.

c. Etiology and pathogenesis
- (1) Etiology. *Listeria* monocytogenes is the causative agent of listeriosis and is commonly found in soil, vegetation, and fecal material.
- (2) Pathogenesis
  - **(a)** Route of **infection.** The neurologic form and the septicemic form rarely occur together.
    - **(i)** The neurologic disease is thought to occur from an ascending infection following a buccal cavity abrasion, with bacteria gaining entry to the brain via the trigeminal nerve.
    - **(ii)** The septicemic **form** of infection is thought to enter the blood via the intestinal tract. Infection may be inapparent with fecal shedding, or septicemia may occur.
  - **(b)** Predisposing **factors**
    - **(i)** Heavy silage feeding. Because L *monocytogenes* can live in spoiled silage (with a pH greater than 5.0), heavy silage feeding is a well-recognized predisposing cause.
    - **(ii)** A sudden weather change to cold and wet, overcrowding, or unsanitary conditions may predispose animals to bacterial invasion. These conditions may also favor a buildup of rotting organic debris, such as silage exposed to air.

d. **Diagnostic** plan and laboratory tests
- **(1)** Clinical signs of focal and lateralizing cranial nerve deficits, such as head tilt, circling, inability to swallow, and facial nerve paralysis, along with depression and pyrexia, are highly suggestive of listeriosis.
- **(2)** CSF analysis. Whereas routine hematology is generally unchanged, a CSF sample has increased protein and leukocytes, typically consisting of small and large mononuclear cells.
- (3) Postmortem examination. The necropsy findings are specific, with **characteristic microabscesses** in the pons and medulla. To culture the organism from the brain, a "cold enrichment" method is necessary. Brain suspensions are held at 4°C and cultured weekly.

e. Therapeutic plan
- (1) Antibiotics. Early treatment with antibiotics such as tetracycline (10–20 mg/kg intravenously every 12 hours for 7 days) or penicillin (44,000 IU/kg every 12 hours for 7–21 days) is clinically effective. Therapy should be continued for at least 7 days after the resolution of signs.
- **(2)** Supportive care during clinical disease is also important with tube-feeding of water and electrolytes to animals that are unable to swallow effectively, and correction of acid–base imbalances that occur with excessive salivary losses.

f. Prognosis The prognosis is fair for complete recovery (50%–75%) if the animal is ambulatory and able to swallow.

g. Prevention
- (1) Management strategies. The only control measures include the isolation of

affected animals and the reduction of silage feeding. In feedlots, constant feeding of low levels of tetracyclines can be considered.

(2) **Human infection.** Because this organism is capable of causing infections in man and is shed in milk and other secretions from cattle, it is particularly important to caution owners of lactating cattle in which the disease has been diagnosed about the risks of consuming unpasteurized milk. Otherwise, Infection of man from infected animals is rare.

4. Brain abscesses

a. Patient profile and history. Brain abscesses can occur sporadically in mature animals of any species, following a pyogenic disease elsewhere in the body. Affected horses are often younger than 3 years. In cattle, there may be a predilection for the pituitary region, with recent placement of rings in the nose in the history.

b. **Clinical findings** include an altered mental status, unilateral or bilateral loss of vision, head turn, compulsive circling, a sluggish or ataxic gait, or other focal cranial nerve deficits, depending on the abscess location. These signs also often fluctuate in severity. In cattle with pituitary abscesses, a dropped jaw and dysphagia are characteristic findings.

c. Etiology and pathogenesis

(1) Etiology

(a) In horses, Streptococcus *equi* and Streptococcus *zooepidemicus* have been cultured from brain abscesses.

(b) In food animals, Actinomyces bovis, Mycobacterium *bovis*, Fusobacterium *necrophorum*, and Actinomyces pyogenes have been isolated from brain abscesses.

(2) Pathogenesis

(a) Although S. *equi* and S *zooepidemicus* are commonly involved with the respiratory tract, infection can result in metastatic abscessation of other organs (e.g., the brain and liver) or *mesenteric* abscesses.

(b) Placement of nose rings in bulls has resulted in a higher incidence of brain abscesses in some situations, possibly because of the bacteria gaining ready access to blood flow in the head via wound contamination at the nose ring site.

d. Diagnostic plan and laboratory tests. The diagnosis is often based on clinical signs and neurologic examination.

(1) Routine hematology is unlikely to reflect inflammation unless septic foci are present elsewhere in the body.

(2) **CSF** analysis often reveals slightly elevated protein levels and the presence of inflammatory cells of a mixed population. However, these changes are not diagnostic for brain abscess.

(3) Ancillary diagnostic tests include cerebral angiography, visual evoked potential recordings, and computed tomography (CT) scans. However, these tests are not accessible at most veterinary facilities.

e. Therapeutic plan. Surgical drainage and appropriate, **prolonged** antimicrobial therapy has been successful in treating brain abscess in horses, but this procedure requires a CT scan for diagnosis and anatomical landmarks for drainage. Antimicrobial therapy alone has not been successful in treating cerebral abscesses in large animals.

5. **Pseudorabies (Aujeszky's** disease)

a. Patient profile. Cases of pseudorabies have been documented in all farm animal species, with the exception of horses. The main carrier animal is the pig. Dogs can also be infected. Although it occurs in some regions of the United States, pseudorabies is exotic to Canada.

b. Clinical findings

(1) In ruminants, signs include intense local pruritus with violent licking or rubbing of the face, limbs, or trunk, accompanied by maniacal behavior (e.g., excitement, circling, vocalization, salivation), ataxia, paralysis, and death within several days. Sudden death can also occur.

(2) In pigs, the infection may be asymptomatic in adults or cause abortions. In piglets, a high mortality rate is associated with neurologic signs.

c. Etiology and **pathogenesis**

(1) Etiology. Pseudorabies is caused by a herpesvirus. Infected swine are the source of infection for other animals.

(2) Pathogenesis. The virus enters abraded skin or the upper respiratory mucosa, and spreads to the CNS through cranial or spinal nerves, causing extensive neuronal damage.

d. Diagnostic plan and **laboratory** tests

(1) **Virus isolation** can be used to make a diagnosis in the live pig, but ruminants affected by pseudorabies usually die in 6–48 hours of the onset of clinical signs.

(2) **Pathologic** findings consist of a nonsuppurative meningoencephalomyelitis.

(3) The histologic findings in the CNS include perivascular cuffing and focal necrosis in the gray matter. Intranuclear inclusion bodies may be found in degenerating neurons.

(4) **Confirmation** of the infection is by virus isolation from postmortem tissue.

e. Differential diagnoses

(1) In **ruminants,** differential diagnoses should include rabies and listeriosis.

(2) In pigs, Teschen disease, salt poisoning, hog cholera, streptococcal meningitis, and African swine fever should be considered.

f. Therapeutic plan. There is no effective treatment, but hyperimmune serum given to baby pigs may help during an outbreak in a swine herd.

g. Prevention

(1) Vaccination. The present control method in the United States is through vaccination, but the vaccine's effectiveness has not yet been evaluated.

(2) A test and removal system can eliminate the disease from a herd of swine. To prevent exposure to other species, infected and uninfected animals should not be housed together.

6. Rabies. Although rabies is rare in most regions, it is of chief concern because of public health considerations.

a. Patient profile. This disease may occur in all warm-blooded animals. Among large animals, cows are more commonly affected than horses, pigs, sheep, or goats.

b. **Clinical** findings. The only typical finding in rabies is its atypicalness.

(1) The mild or paralytic form shows variable signs consistent with progressive ascending paralysis, including knuckling of the hind fetlocks; sagging and swaying of hindquarters; decreased sensation over the hindquarters; paralysis of the tail, anus, and penis; dribbling urine; salivation; recumbency; and eventually death.

(2) The **furious** form occurs if the virus reaches the forebrain and is characterized by tenseness and hypersensitivity to sounds and movement. Affected animals may attack inanimate objects or other animals, bellowing loudly and purposelessly, or they may show sexual excitement.

c. Etiology and pathogenesis

(1) Etiology. Rabies is caused by a rhabdovirus.

(2) Pathogenesis

(a) Transmission. Rabies is transmitted through bite wounds after localizing in the sensory end organs of the olfactory nerve of the infected host carnivore. The principal reservoirs in North America are wild carnivores (e.g., skunks, foxes, raccoons), whereas the vampire bat is a key reservoir for the virus in South America. Rabies is also enzootic in insectivorous bats in Western Canada.

(b) **Route** of **infection.** The virus spreads from the site of the infection to the CNS via peripheral nerves. The incubation period is 2 weeks to several months, depending on the bite site.

d. Diagnostic plan and **laboratory** tests. Rabies is extremely difficult to diagnose and should be considered when dealing with any obscure neurologic disease. If

rabies is suspected, any diagnostic sampling or animal handing should be performed with caution.
- (1) The animal can be treated for any other possible treatable diseases, such as lead toxicosis or metabolic disease, which may also alter behavior.
- (2) If rabies is highly suspected, the regulatory authorities should be contacted regarding current guidelines for animal handling. Affected animals may be evaluated for a period of 10 days.
- (3) Postmortem examination. If death ensues, the brain and fresh salivary gland tissue should be submitted to an appropriate laboratory for further diagnostic testing. Half the brain should be fresh and the other half fixed in formalin.
  - (a) Fluorescent antibody testing and mouse inoculation studies are used for confirmation of the diagnosis.
  - (b) Histologic examination of the brain for Negri bodies is also used but can yield some false-positive results.
  - (c) The intracerebral inoculation of weaned mice with brain tissue is highly soecific for infection, but this can take at least 3 weeks for confirmation of infection.
- e. Therapeutic plan. There is no treatment for animals showing clinical signs. Anti-inflammatory drugs, which may be used concurrently for other diseases, may delay the progression of signs.
- f. Prevention. Currently, there are inactivated diploid vaccines that are licensed for all veterinary species. These vaccines appear to be highly protective. In regions where rabies is endemic, horses and other valuable farm animals should be vaccinated.

7. Equine viral encephalitis
   a. Patient profile. All ages and breeds of horses are susceptible, but the disease is not common in suckling foals younger than 3 months. Equine encephalitis occurs in late spring and summer because of the transmission by insect vectors. However, in the southeastern part of the United States, infection can occur any time of the year.
   b. Clinical findings
      (1) The disease may be subclinical or may appear in the septicemic form with pyrexia, anorexia, and depression. With encephalomyelitis, there is fever, anorexia, and severe depression.
      (2) Other neurologic changes may include dementia with blindness, head pressing, ataxia, weakness, and seizures. Severely affected horses can become recumbent and have respiratory arrest.
   c. Etiology and pathogenesis
      (1) Etiology. The arboviral encephalitides (eastern, western, and Venezuelan) in horses are classified as alphaviruses of the togavirus family.
         (a) Eastern equine encephalitis (EEE) is most prevalent in the southeastern United states.
         (b) Western equine encephalitis (WEE) is usually restricted to western Caw ada and the western United States. WEE is less severe than EEE.
         (c) Venezuelan equine encephalitis (VEE) occurs outside North America and has far higher morbidity and mortality rates.
      (2) Pathogenesis
         (a) Transmission. The diseases are transmitted to horses by a mosquito vector.
            (i) Equine infection. Horses are considered a "dead-end" host, with infected birds being the reservoir hosts for these viruses.
            (ii) Human infection. The equine population acts as a sentinel animal, with equine cases preceding human cases by 2–5 weeks. Human cases tend to be "flu-like," with young and old people susceptible to encephalitis.
         (b) The viruses are neurotropic and cause direct neuronal necrosis throughout the entire CNS, particularly in the cerebrum, causing the characteristic severe mental depression.

- d. Diagnostic plan and laboratory tests. Diagnosis of EEE and WEE is based on signs, season, locale, and serum titers.
  - (1) CSF analysis reveals increased protein and WBC counts. Neutrophils are present in the acute disease, but mononuclear cells predominate in later stages. A slight xanthochromia may also be present.
  - (2) Serologic titers can be measured by hemagglutination inhibition (HI) or complement fixation (CF). A fourfold rise in titer in samples collected 7–10 days apart or a very high single titer in an unvaccinated animal (1:160 for EEE and 1:40 for WEE) are diagnostic. Additionally, the use of immunoglobulin M (IgM)-specific enzyme irnmunoassays can help in the antemortem diagnosis of EEE.
- e. Therapeutic plan. The prognosis is grave for EEE and poor to fair for WEE.
  - (1) Supportive and symptomatic care. If the animal remains ambulatory, it should be given supportive and symptomatic therapy. This includes force feeding and fluid therapy because the depression is often so severe that the animal will leave food in the mouth, although swallowing is not impaired.
  - (2) Therapeutic agents. If depression progresses and pupils become unresponsive to light, cerebral edema may be developing.
    - (a) Mannitol (20%) may be given intravenously at 0.25–0.5 g/kg.
    - (b) The use of dexamethasone is controversial, but in the initial stages, it may be useful at a dose of 0.1–0.25 mg/kg.
    - (c) DMSO (1 g/kg in a 10% solution of 5% dextrose) is probably the drug of choice for treating cerebral edema.
- f. Prevention. Vaccination for EEE and WEE is the best preventive method. The animals should receive two injections 3–4 weeks apart 1 month before mosquito season. Semiannual vaccinations may be necessary where mosquito seasons are long. Fortunately, there is no transmission from horse to man, and even the infected hone does not pose a significant risk to other horses.

8. Yellow star thistle or Russian knapweed toxicity
   a. Patient profile. This toxicosis is seen in the northwest United States or Australia in late summer or autumn and affects any age, gender, or breed of horse.
   b. Clinical findings. Affected horses exhibit an acute onset of rigidity of the mastication muscles. This rigidity results in a characteristic grimacing expression, with the mouth held half open and the lips and nostrils drawn back. Protrusion of the tongue may be present. The difficulty in prehension may progress to a complete inability to eat or drink.
   c. Etiology and pathogenesis
      (1) Etiology. Disease is caused by the ingestion of yellow star thistle (Centaurea solstitialis) or Russian knapweed (Centaurea repens).
      (2) Pathogenesis. The plant does not appear to be toxic to ruminants but is thought to contain an antimetabolite that is specifically toxic to horses. Ironically, sheep thrive on a diet consisting solely of these plants.
   d. Diagnostic plan and laboratory tests
      (1) Classic clinical signs and a history of possible exposure to these plants form the basis for diagnosis. There are usually no significant alterations of diagnostic value on laboratory analysis.
      (2) Necropsy. The major lesion at necropsy is in the brain. It is usually bilateral and consists of areas of necrosis in the globus pallidus and substantial nigra (encephalomalacia).
   e. Therapeutic plan and prognosis. There is no cure for this disease, but the animal can be supported via nasogastric tube feeding. However, the prognosis is grave, and the animal usually dies of dehydration or starvation if no nutritional support is given.
   f. Prevention. Horses should be kept from pastures that contain these toxic plants, but sheep or cattle can graze these pastures safely.

9. Ammoniated feed toxicity ("bovine bonkers, bovine hysteria")
   a. Patient profile. This disease is seen in adult cattle, calves, and sheep that are

given ammoniated feed. Often, a new batch of urea-containing feed has been fed to the animals.

b. Clinical findings .

(1) Clinical signs include acute staggering, recumbency, and even death. Frequent urination and defecation, trembling, sweating, and ear twitching are often observed.

(2) Physical examination. Bloat, dyspnea muscle tremors, and abdominal pain are found on physical examination. On neurologic examination, there is incoordination and blindness, accompanied by hyperexcitability, hyperesthesia, restlessness, colliding with inanimate objects, seizures, and coma.

c. Etiology and pathogenesis. Affected animals have consumed excessive urea in their feed to which their rumen may not be accustomed. Urea is converted to ammonia in the rumen and liver, with alkalosis and ammonia encephalopathy resulting.

d. Diagnostic plan and laboratory tests. An elevated blood and CSF ammonia concentration in the absence of liver disease is diagnostic for urea or ammonia intoxication.

e. Therapeutic plan. Rumen lavage and evacuation is necessary to reduce the ongoing absorption of the urea. Following lavage, acidifying solutions (such as acetic acid) along with cold water should be given orally to help counter the systemic alkalosis.

f. Prognosis. The prognosis is guarded to grave. When clinical signs are obvious, the disease is usually fatal.

g. Prevention. The gradual introduction of feed containing urea should prevent this disease.

10. Hypovitaminosis A

a. Patient profile. This disorder can affect most young growing animals and is also found in adult cattle.

b. Clinical findings

(1) Calves. Experimental and naturally occurring vitamin A deficiency in calves results in blindness, ill thrift, diarrhea, and pneumonia. The blindness in calves is peripheral, with absent pupillary light reflexes and dilated pupils in severe cases.

(2) In adult cattle, in addition to blindness, there may be convulsions, diarrhea and generalized edema.

(3) Young horses that are fed vitamin A-deficient diets have shown night blindness, ill thrift, and seizures, but naturally occurring disease in horses is rare.

c. Etiology and pathogenesis. Vitamin A is necessary for normal bone growth and epithelial maintenance. The pathophysiology appear to be the same in all species, but clinical signs differ according to skull anatomy.

(1) Brain distortion and herniation. In the CNS, a lack of vitamin A distorts cranial bone development and retards endochondral ossification, resulting in brain distortion and herniation.

(2) Seizures. Reduced CSF absorption across arachnoid villi leads to increased CSF pressure, which may manifest clinically as seizures.

(3) Blindness can occur from optic canal restriction, with optic nerve entrapment in the optic foramina.

d. Diagnostic plan and laboratory tests. Serum vitamin A concentrations are usually found to be low (2–15 g/dl).

e. Therapeutic plan. Affected animals should be given vitamin A (440 IU/kg parenterally). There is usually a dramatic response to treatment within 48 hours, with the exception of prominent blindness, which is not likely reversible.

f. Prevention. Current management practices include feeding adequate vitamin A. By merely increasing green feed a hay quality, dietary vitamin A is usually sufficient. Additionally, animals can be given injectable repositol forms of vitamin A.

E. Disorders accompanied by multiorgan involvement

1. Scrapie

a. Patient profile. This long-recognized disease affects adult sheep between the ages of 2 and 5 years and also goats. Scrapie occurs more readily in some sheep breeds that have genetic predilection for the infection.

b. Clinical findings. The main signs are cerebellar incoordination and pruritus.

(1) There are transient episodes of nervousness, behavior changes, emaciation, seizures, and recumbency. Eventually death occurs.

(2) A fine head tremor is often noted. Pruritus is severe, with a characteristic head elevation and nibbling reaction when the skin is scratched.

c. Etiology and pathogenesis

(1) Etiology. The causative organism is a small protein unit (i.e., viroid).

(2) Pathogenesis

(a) Transmission. Scrapie is classified as a "slow virus" infection, with vertical or horizontal transmission and a prolonged incubation period of months to years.

(b) Route of infection. The viroid replicates in lymphoid tissue and invades the central nervous tissue, producing a spongiform encephalomyelopathy with neuronal vacuolation and the degeneration of cerebrospinal, cerebellospinal, and optic nerve tracts.

d. Diagnostic plan. There is no accurate antemortem method to screen for infected animals, and diagnosis is based on postmortem examination of the brain tissue.

e. Therapeutic plan and prevention. There is no treatment, and affected animals should be slaughtered because the disease is invariably fatal. Scrapie has been a reportable disease but was recently removed from this designation in various jurisdictions.

2. Bovine spongiform encephalopathy (BSE, mad cow disease). As a consequence of its association with spongiform encephalopathy (Creutzfeldt-Jakob disease) in humans, BSE is currently the cause of a large-scale slaughter of adult cattle in Britain and other European countries. The United States and Canada have imposed strict guidelines for the import of live animals in attempts to ensure that these countries remain free of the disease.

a. Patient profile. This disease affects adult cattle and was first described in Great Britain.

b. Clinical findings. Affected cattle exhibit apprehension, mild incoordination, hyperesthesia that progresses to severe behavioral changes, recumbency, and death.

c. Etiology and pathogenesis

(1) Etiology. BSE is thought to be caused by a "scrapie-like" agent. The causative agent appears to have entered the British cattle population through a change in feed processing, which facilitated the introduction of sheep offal into animal feeds in the early 1980s. This allowed exposure to a "scrapie-like" agent, which, because of the long incubation period, became manifest years later.

(2) Pathogenesis. The infection of the brain tissue by this agent causes a spongiform encephalopathy, but the mechanism is not completely understood.

d. Diagnostic plan. Postmortem microscopic examination of the brain is the only known method to determine the diagnosis.

e. Therapeutic plan and prevention. There is no treatment, and the slaughter of all animals at risk and their offspring has been implemented.

3. Hepatic encephalopathy (see also Chapter 5 I B 1 b) occurs in animals with acute or chronic liver failure. Most species and ages can be affected in acute or chronic liver failure, however, affected animals are usually adults. Clinical findings include depression, head pressing, and yawning that may wax and wane, depending on the

time since the last feeding. Hepatic encephalopathy must be differentiated from primary neurologic diseases that may also affect behavior and mental activity.

4. **Leukoencephalomalacia** (moldy corn disease)
   a. Patient profile and history. This disease occurs in horses of any breed, sex, or age that have a history of ingestion of moldy corn. Outbreaks can occur.
   b. Clinical findings. Affected animals have a sudden onset of dementia, blindness, convulsions, or all of these signs. Sudden death can also occur. Additional neurologic findings can include circling, excitability, drowsiness, and ataxia in various combinations and possibly asymmetric findings.
   c. Etiology and **pathogenesis**
      (1) Etiology. A toxin, likely fumonisin, produced by *Fusarium moniliform* in moldy corn has been indicated as the causative agent.
      (2) Pathogenesis. After ingestion of the toxin, horses develop liquefactive cerebral necrosis and associated brain swelling, which may result in herniation and brain stem compression. Toxic hepatopathy can also develop and may contribute to the clinical signs.
   d. Diagnostic plan and laboratory tests
      (1) The clinical signs, neurologic findings, and history of feeding moldy corn are used as a basis for diagnosis. Finding moldy corn alone does not confirm a diagnosis because up to 80% of corn samples can contain *Fusarium* species.
      (2) **CSF** analysis may be normal or may show a pleocytosis with elevated neutrophils and protein.
   e. Therapeutic plan. The only suggested treatment is to remove **horses** still at risk from access to the moldy feed and give oral cathartics to those exposed. **Glucocorticoids** or DMSO for presumed brain swelling can be given to clinically affected horses.
   f. Prognosis. For mildly affected horses, the prognosis for recovery is good, with little or no residual brain damage. Most horses, however, die shortly after the onset of clinical signs.

III. SPINAL CORD DISEASES

A. Equine

1. Cervical vertebral **malformation (CVM;** wobbler disease, wobbles, cervical **stenotic myelopathy)**
   a. Patient profile and history
      (1) Patient profile. This disease is found in young horses and usually occurs when the animal is less than 2 years old. There appears to be a sex predilection, with colts being affected more than fillies. Thoroughbreds appear to have the highest incidence.
      (2) History. The onset of signs can be sudden and may be related to a traumatic incident, or it may be insidious. Animals are usually in good bodily condition and on a good plane of nutrition.
   b. Clinical findings. Clinical signs are referable to upper motor neuron **(UMN) problems** of both the front and hind limbs, suggesting a lesion in the cervical spinal cord.
      (1) Ataxia. The animals exhibit varying degrees of ataxia.
         (a) The hind limbs are usually more severely affected than the rear, but the signs are usually symmetric from left to right.
         (b) There can be toe dragging at the walk and rear limb **circumduction** when the horse is led in a tight circle.
         (c) **Proprioceptive** deficits are exaggerated when the animal is forced to negotiate a hill, turn sharply backward, or step over obstacles.
      (2) Paresis may be evident in response to downward pressure over the withers

and loins and when pulling the horse to the side by its tail while at the walk (sway test).
      (3) Hypermetria and spasticity may also be seen to a lesser degree than the ataxia. When left alone, the animal may assume awkward positions (e.g., legs extended to the side, legs crossed).
   c. Etiology and pathogenesis
      (1) Etiology. No specific cause has been identified for CVM. One school of thought favors genetics as a cause, whereas others feel the disease is a result of rapid growth combined with high energy nutrition or nutritional imbalances. Probably both of these factors are involved.
      (2) Pathogenesis. Classically, horses suffering from CVM were called "wobblers." A wobbler is a broad term that describes a set of clinical signs that may be caused by several different disease processes. Two forms of CVM have been recognized: a functional (dynamic) stenosis and an absolute (static) stenosis. Both forms occur between the ages of weanling and 3 years, with functional stenosis appearing at a somewhat younger age.
         (a) In functional stenosis, spinal cord compression is caused by instability between cervical vertebrae. When the neck is placed in a flexed or hyperextended position, the vertebrae subluxate into the spinal canal.
         (b) In absolute stenosis, there is spinal cord compression that is not altered by the positioning of the neck. Osseous changes of the cranial articular processes and medial ingrowth of the articular processes can cause this stenosis.
   d. Diagnostic plan and laboratory tests
      (1) The neurologic examination is suggestive of a focal cervical lesion for which cervical radiographs should be taken. Remodeling of the caudal vertebrae epiphysis and the articular facets may be seen in functional stenosis.
      (2) A **myelogram** is essential for a positive diagnosis of spinal cord compression, which commonly occurs at C3–4 and C4–5 (for **functional** stenosis) and at C5–6 and C6–7 (for absolute stenosis).
      (3) Routine laboratory samples, such as a CBC and biochemistry panel, are of no diagnostic value for **affected** horses.
      (4) **CSF** analysis, although useful to rule out other diseases causing similar signs, is usually normal.
   e. Differential diagnoses. Each of the following disorders can be ruled out by its corresponding method of positive diagnosis.
      (1) Trauma—myelogram, CSF analysis, history
      (2) Equine protozoal **myelitis**—CSF analysis, asymmetry, lower motor neuron (LMN) involvement
      (3) Herpes I myelitis—titer, recovery
      (4) Equine degenerative myeloencephalopathy—postmortem examination
      (5) Cerebrospinal **nematodiasis**—CSF analysis
      (6) Space-occupying lesion (**neoplasia, abscess**)—CSF analysis
   f. Therapeutic plan
      (1) Medical treatment with anti-inflammatory drugs plus stall rest may arrest the progress of clinical signs for some time, but usually the disease progresses. Additionally, there are ethical questions about treating such cases for which complete recovery is unlikely if the horse is likely to be ridden at high speeds.
      (2) Surgical treatment includes arthrodesis of the unstable vertebrae in cases of functional stenosis or laminectomy to aid in decompression of absolute stenosis. The arthrodesis technique may halt the progress of the ataxia and even allow some return to usefulness.
   g. Prognosis. The prognosis for recovery is guarded to grave. Surgical correction is expensive, and its application to an equine athlete is controversial.

2. Equine degenerative **myeloencephalopathy (EDM).** First described in 1977, EDM may now account for 25%–50% of ataxic horses seen at referral clinics in North America.

a. Patient profile. EDM is an ataxia that affects horses less than 12 months old, with the oldest affected animal about 3 years of age. No breed or sex determination has been noted, and the signs, when observed, can be progressive or stabilize.

b. Clinical findings
   (1) Affected horses exhibit symmetrical ataxia, with marked proprioceptive deficits when asked to circle or turn. Paresis and spasticity may also be noted.
   (2) In contrast to CVM, the front limbs may have more severe ataxia or paresis than the rear limbs or may be only minimally affected in combination with profound pelvic limb changes, which is not consistent with focal cervical compression.

c. Etiology and pathogenesis
   (1) Etiology. The etiology of EDM is unknown but is thought to be related to inappropriate vitamin E levels or metabolism in some horses. The roles of hereditary, congenital, toxic, and nutritional factors have not been determined, but the disease resembles heretodegenerative diseases in humans. The disease appears to be associated with increased dependence on hay as the sole diet and with animals that are not allowed much grazing time on fresh forage.
   (2) Pathogenesis. The neurohistopathology of EDM indicates a degenerative process throughout the nervous tissue that is more pronounced in the thoracic segments of the spinal cord. There is diffuse neuraxonal degeneration of nuclei in the brain. Clinically, the signs are restricted to symmetrical abnormalities in the posture and gait of all four limbs.

d. Diagnostic plan and laboratory tests
   (1) Clinical signs and age may suggest EDM, but there is no antemortem method to differentiate patients from other "wobblers," except by elimination of other causes of ataxia.
   (2) Cervical radiography is normal in affected animals.

e. Differential diagnoses. The main differential diagnosis is cervical vertebral malformation.

f. Therapeutic plan. No curative treatment is available, and signs will plateau. However, high doses of vitamin E have been shown to be effective in preventing the progression of clinical signs and may play a role in prevention in animals at risk.

g. Prevention. Supplemental dietary vitamin E (up to 6000 IU/day) can be given to related young horses at risk. Because there may be a genetic factor involved, selective breeding may be advisable.

3. Equine protozoal myeloencephalitis (EPM)
   a. Patient profile and history
      (1) Patient profile. This spinal disease affects horses ages 2–7 years, particularly Standardbreds and Thoroughbreds. EPM has been frequently noted in racing and breeding animals, with males and females equally affected. Serologic evidence suggests widespread exposure to this parasite in the eastern and Midwestern United States, and it is likely that most horses that are seropositive have mounted an immune response and cleared the organism before it reached the CNS.
      (2) History. EPM may appear as a mild lameness in the hind limbs that has defied localization.
   b. Clinical findings. Signs may occur suddenly or insidiously.
      (1) Clinical signs can be varied, with a history of ataxia, stumbling, falling while being transported, or notable limb weakness. Gait abnormalities may be asymmetric. Mild cases may manifest only as ill-defined hind limb lameness.
      (2) Muscle atrophy of isolated muscle groups, most commonly noted in the gluteals, suprascapular, and masseter areas or tongue, have been identified as being highly suggestive of EPM because the disease can result in LMN deficits.

c. Etiology and pathogenesis
   (1) Etiology. EPM is associated with a coccidian parasite, *Sarcocystis* falcatula.
   (2) Pathogenesis. *S. falcatula* is a parasite that has an obligate two-host life cycle; the definitive host being opossums, and the intermediate host being birds. The disease is sporadic but can occur in several animals in the same locale, and the incidence of disease is said to follow closely the geographic range of opossums.
      (a) The parasite encysts in the muscle of birds and when this tissue is eaten by opossums, the organism undergoes sexual reproduction in the intestinal epithelium and is shed as infective oocysts in the feces.
      (b) Horses are an aberrant intermediate host for the parasite. Sporocysts are ingested and although tissue cysts do not develop, some of the parasites spread as tachyzoites to the CNS, where they continue to undergo asexual reproduction in neurons and microglial cells. Because cysts do not form, horses cannot transmit *S.* falcatula to other animals.

d. Diagnostic plan and laboratory tests. Diagnosis is based on clinical signs, response to treatment, and recently, analysis of CSF for the presence of antibodies. A CBC and biochemistry panel are invariably of little value.
   (1) Clinical signs of an asymmetric ataxia with LMN involvement may aid in a diagnosis, but they may not be present.
   (2) Response to treatment. A response to empiric therapy with an antiprotozoal agent [see III A 3 e (1)] aids in differentiating EPM from other spinal cord disorders.
   (3) CSF analysis
      (a) Presence of **antibodies.** Recently available serologic tests can provide evidence of exposure to this protozoan parasite. Furthermore, detection of antibodies in the CSF is highly suggestive of intrathecal production of antibodies as a result of nervous tissue infection. However, any blood contamination of the CSF sample can give a false-positive result in a serologically positive horse.
      (b) Presence of *S. falcatula* DNA. Testing the CSF for the presence of the parasite's DNA can also be done and provides a highly sensitive test for early EPM infections.
      (c) **Other** findings. Mild xanthochromia, a slight increase in protein level (80–100 mg/dl), and a moderate pleocytosis (10–100 leukocytes/μl, consisting primarily of mononuclear cells) may be noted.
   (4) Postmortem findings. Gross necropsy findings may include focal areas of hemorrhage or discoloration in the brain and spinal cord. A multifocal, necrotizing, nonsuppurative myeloencephalitis is seen. Protozoal organisms may be seen in 50% of the cases.

e. **Therapeutic** plan. Some animals may respond quickly within a few weeks, whereas others may require months of treatment. There may also be no response to therapy.
   (1) **Antiprotozoal** treatment
      (a) By extrapolation from the treatment of toxoplasmosis in humans, affected horses have been treated successfully using long-term medication with **trimethoprim-sulfadiazine** (15 mg/kg twice a day), combined with pyrimethamine (0.25 mg/kg orally every 24 hours). Both of these drugs impair folic acid metabolism.
      (b) When treating with folic acid antagonists, frequent **hemograms** should be evaluated because of a reduction in hematopoiesis. **Folic** acid supplementation (e.g., by adding brewer's yeast to the feed) may be needed if thrombocytopenia or anemia becomes a clinical problem.
   (2) Corticosteroids are contraindicated because of their immunosuppressive properties.

f. **Prognosis.** The prognosis for recovery is fair to good (50%–75%) with appropriate treatment, but depends considerably on the severity and duration of clinical signs when the disease is first identified. A good prognostic indicator is a rapid response to antiprotozoal treatment in the first 2 **weeks.**

4. Equine herpesvirus 1 (EHV-1) myeloencephalitis
   a. Patient profile and history
      (1) **Patient profile.** Horses of any age, breed, or sex can be affected.
      (2) **History.** There may be a recent history of upper respiratory tract infection (see Chapter 6 II A 3 b) or abortion problems on the farm.
   b. Clinical findings
      (1) **Ataxia.** There is usually an acute onset of weakness and ataxia (in one or all limbs) or even recumbency. Usually the hind limbs are symmetrically affected. These signs may vary in severity from slight involvement to peracute tetraplegia, which quickly stabilizes.
      (2) **Fever** at 39°C–40.5°C may be present, along with distal **limb edema.**
      (3) **Urinary incontinence,** bladder distention, and **hypotonia of the anus** and tail may be noted.
      (4) Weakness of the tongue, jaw, or pharynx and vestibular signs can also occur, but are less common.
   c. Pathogenesis. **EHV-1 myeloencephalitis** may be an immune complex disease.
      (1) EHV-1 infection causes abortion and neurologic disease possibly because of its tropism for endothelium.
      (2) The myeloencephalitis is primarily a vasculitis that affects both the gray and white matter of the brain and spinal cord.
   d. Diagnostic plan and laboratory tests
      (1) CSF analysis. The CSF often has a markedly xanthochromic appearance, with elevated protein levels (100–500 mg/dl), but cell counts are generally within normal limits. These findings are highly suggestive of the vasculitis.
      (2) Serology. Paired serum titers to EHV-1 showing a fourfold rise is good circumstantial evidence of EHV-1 involvement. Preexisting EHV-1 serum-neutralizing titers do not appear protective and may even be required for the neurologic form of the disease.
   e. Differential diagnoses. The differential diagnoses are the same as those for CVM (see III A 1 e).
   f. Therapeutic plan
      (1) Stall rest. Slightly **affected** animals that remain standing usually recover uneventfully with 2–3 weeks stall rest. Recumbent animals are less likely to re cover.
      (2) Extensive nursing care is needed to prevent secondary complications of pressure sores, cystitis, and hypostatic lung congestion. Heavy bedding, sterile urinary catheterizations, and frequent rolling of the animal are necessary for success. If practical, slinging may help.
      (3) Corticosteroids. The use of corticosteroids has been suggested by some authors because the process in the CNS is thought to be immune mediated. However, corticosteroid administration is controversial because of the drug's immunosuppressive effects and particularly because EPM may be a possible diagnosis.
   g. Prognosis. For the mildly affected animal, the prognosis for recovery is good.
   h. Prevention, Vaccination against EHV-1 may be beneficial, but the efficacy of vaccination against the neurologic form has not been proven.

5. Cerebrospinal **nematodiasis**
   a. Patient profile. This disease occurs sporadically, affecting any age, breed, or sex.
   b. Clinical findings. Ataxia, weakness, and head signs vary with migratory sites. Affected horses may be depressed and anorectic.
   c. Etiology and pathogenesis. Several parasites have been found in the CNS in animals with this disease, including *Strongylus vulgaris, Hypoderma* species, *Setaria* species, Draschia species, *Micronema* species, and *Parelaphostrongylus* species. Malacic tracts are found along the migratory path. The cause of these aberrant migrations is not known.
   d. Diagnostic plan and laboratory tests. The main method of diagnosis is finding elevated eosinophil counts in the CSF. Other laboratory tests are generally nondiagnostic.

   e. Therapeutic plan
      (1) Thiabendazole. Suggested treatments of a 10-times the normal dose of thiabendazole for 2 consecutive days has been suggested, but this drug is no longer commercially available.
      (2) **Ivermectin** can be given but is unlikely to cross the blood–brain barrier to reach the larvae.
      (3) **Corticosteroids** should be given in conjunction with a larvicide drug to decrease inflammation after killing the parasites (0.1 mg/kg dexamethasone every 12 hours for 2 days).
   f. Prognosis. As in other spinal cord diseases, the prognosis for recovery is guarded, and the long-term use of the horse for riding should not be expected.

B **Food** animal

1. Caprine arthritis-encephalitis (CAE; see also Chapter 13 III D)
   a. Patient profile. This disease affects young goats, usually between the ages of 1 and 4 months, but occasionally is seen in adults. Often, more than one kid is affected over a period of time.
   b. Clinical findings. The incidence of subclinical disease is high. Approximately 80% of clinically normal animals are positive on agar gel immunodiffusion (AGID) test for CAE.
      (1) In kids affected with the neurologic form, there is ascending paresis and ataxia, usually beginning in one or both hind limbs and progressing to tetraplegia. The animal is bright, alert, and responsive. Spinal reflexes remain intact, which indicates UMN disease. Mild LMN signs (such as hyporeflexia, blindness, head tilt, and facial nerve paralysis) may occur as the disease progresses. The clinical course of the disease can be 7–14 days.
      (2) In adults, infection generally results in chronic arthritis. Joint distention and hard udders may be seen in adults belonging to affected herds.
      (3) In the arthritic form, there is chronic polyarthritis in adult goats (see Chapter 13).
   c. Etiology and pathogenesis
      (1) Etiology. This disease is caused by the CAE virus, a retrovirus that is antigenically similar to the Maedi-visna viruses in sheep.
      (2) Pathogenesis. Perinatal horizontal spread results from close contact, particularly from colostrum and milk.
   d. Diagnostic plan and laboratory tests
      (1) Serological tests
         (a) CSF samples show a mononuclear pleocytosis with protein elevation.
         (b) AGID test. Serum can be tested for presence of antibody to CAE by the AGID test. This is useful for testing exposure in a herd, but is of little diagnostic value for an individual animal.
      (2) Postmortem **findings.** At necropsy, the neural lesions are mainly restricted to the white matter. Marked demyelination, perivascular cuffing with mononuclear cells, and parenchymal infiltration with macrophages are seen on histopathology.
   e. Therapeutic plan. There is no effective treatment for the ascending paralysis.
   f. Prevention. Control of the disease may be accomplished by separating infected animals from the herd. The CAE virus is passed to the kids through colostrum and milk; therefore, newborn kids should be separated from the dam at birth and fed a safe source of colostrum or pasteurized milk.

2. Swayback (enzootic ataxia)
   a. Patient profile. This disease occurs in newborn sheep, growing lambs, kids, and possibly piglets.
   b. Clinical findings. The course of the disease depends on the age of the animal at presentation.
      (1) Newborn animals may be born dead or weak, or they may develop a progressive, ascending paresis and ataxia. Newborns usually die in 3–4 days.
      (2) Older animals that are affected may live for 3–4 weeks. LMN signs of

hypotonia, muscle atrophy, and depressed reflexes are present and highly suggestive of the disease. Animals ages 3–12 weeks show progressive ataxia of the pelvic limbs.

  (3) Sheep exhibit weakness, recumbency, and blindness. Ewes are unthrifty, anemic, and have a poor fleece. Diarrhea, spontaneous fractures, and acute death also may be seen in ewes.

 c. Etiology and **pathogenesis**
  (1) **Etiology.** Swayback is the result of copper deficiency, which may cause hypomyelinogenesis and porencephaly in utero.
  (2) Pathogenesis
   (a) Swayback develops in lambs, kids, and piglets that are born to dams that either have a copper deficiency or an increase in copper antagonists, such as molybdenum, sulfate, and cadmium.
   (b) **Resulting** neurologic damage
    (i) Cavitations of the cerebral **white** matter, chromatolysis, and **myelin** degeneration in the brain stem and spinal cord may be found on necropsy.
    (ii) At the cellular level, neuroaxonal defect, myelin degradation, and death of the neuron cell body are found.

 d. Diagnostic plan and laboratory tests
  (1) The neurologic findings of ataxia and weakness with LMN changes in lambs, kids, or piglets are highly suggestive of swayback.
  (2) Blood and tissue copper levels confirm the diagnosis. Unaffected herd mates should also be sampled to determine copper levels of the whole farm.
  (3) CSF samples are normal.

 e. Differential diagnoses include CAE, listeriosis, and cerebrospinal parelaphostrongylus.
 f. Therapeutic plan. Copper injections may be followed by some improvement but the neurologic deficits are likely permanent.
 g. Prognosis. The prognosis for recovery is very poor.
 h. Prevention. The pregnant animals in the herd should receive injectable copper, or be provided adequate copper in the ration. Orally administered oxidized copper wire or cupric oxide particles, produced for cattle and sheep, may be the most preferred mode of prevention because they provide a sustained supply of blood copper.

3. Vertebral body abscess
 a. Patient **profile** and history
  (1) Patient profile. This disorder occurs most commonly in young farm animals, particularly cattle, sheep, goats, and swine, but it can also occur in foals.
  (2) History. Omphalophlebitis or pneumonia is a common historic or clinical finding. In lambs, these abscesses are commonly associated with the docking of tails.
 b. **Clinical** finding
  (1) Affected animals may be observed to have neck or back pain and ill thrift. There may be a sudden onset of cervical or back pain, with or without progressive paresis.
  (2) If a **pathologic** fracture has occurred, there is paresis, with lesion localization to the site of the abscess. At this site, there may also be heat, pain, or swelling, but it can also be nondetectable externally.
 c. **Pathogenesis.** Septicemic infection in young animals may localize in vertebral bodies, particularly in the thoracic region in calves. Otherwise, any site on the vertebral column can be involved.
 d. Diagnostic plan and laboratory tests
  (1) Clinical signs are suggestive of spinal cord damage.
  (2) A CBC may also be suggestive of an inflammatory process, with a neutrophilia and increased fibrinogen.
  (3) A CSF sample can show evidence of compression, with xanthochromia and mild increases of protein and cell content. However, CSF can also be nor-

mal because the septic process is usually external to the dura of the spinal canal.
  (4) Radiography and myelography may be rewarding when diagnosing young animals.
 e. Therapeutic plan. Euthanasia is indicated for all but the most valuable animals. However, high-dose, long-term antibiotic therapy can be attempted for animals of considerable value if the owner wants to continue treatment.
 f. Prognosis. Recovery is unlikely when clinical signs are obvious, particularly if there is a pathologic fracture.

IV. NEUROMUSCULAR DISEASE

A. Tetanus
 1. Patient profile and **history**
  a. **Patient** profile. All farm animals, including horses, swine, cattle, sheep, and goats, can develop tetanus. Horses are most susceptible to the neurotoxin, and cattle are least susceptible.
  b. History. Affected animals may have a history of sustaining a penetrating wound in the 3 weeks previous to the onset of clinical signs.

 2. Clinical **findings**
  a. Early signs include stiffness, muscle tremors, increased spastic reflex responses to stimuli (e.g., hand clap, head tap), and difficulty chewing, swallowing, or prehending feed.
  b. Later signs. As the disease progresses, the animal takes on a sawhorse stance, with elevated ears and tailhead. The animal exhibits "flashing" of the third eyelid, increased jaw tone, and extreme sensitivity to external stimuli, which can induce violent muscle spasms. Cattle can also show mild bloat
  c. Terminally, the animal falls into lateral recumbency, has opisthotonos, and eventually suffers respiratory failure, causing death. These animals die 5–10 days following the appearance of clinical signs.

 3. Etiology and **pathogenesis**
  a. Etiology. Tetanus is caused by the toxins of *Clostridium* tetani, a gram-positive facultative to obligate anaerobe.
  b. Pathogenesis
   (1) Route of infection
    (a) Tetanus organisms (i.e., spores) are in the animal's environment and usually enter the body through penetrating or contaminated wounds (e.g., retained placenta, puncture wounds to the foot in hones, elastrator bands for tail docking or castration).
    (b) The wound site must have some tissue trauma and lack oxygen for the spores to grow. Signs can occur any time from 1–3 weeks post injury or occasionally longer.
    (c) In **ruminants,** it is postulated that toxin may be produced in the gastrointestinal tract.
   (2) Neurotoxin migration. C. tetani produces a neurotoxin that migrates along nerve fibers and in blood and lymph fluid from its site of production. This neurotoxin acts on four regions of the nervous system: the motor end plate of skeletal muscle, the inhibitory interneurons of the spinal cord, the brain, and the sympathetic nervous system. The inhibitory interneurons predominately involve the antigravity (extensor) muscles; thus, the signs of tetanus reflect the overactivity of these muscles.

 4. Diagnostic plan. The diagnosis is made on the clinical signs of hyperesthesia to external stimuli. Demonstrating circulating neurotoxin is not feasible and isolation of the organism from contaminated wounds is difficult

5. Differential diagnoses may include hypocalcemic tetany of mares, hypomagnesemic tetany of cattle and calves, acute laminitis of horses, or PEM of cattle.

6. Therapeutic plan

a Tetanus antitoxin (TAT). Toxin neutralization using TAT is a key part of treatment. Although TAT cannot penetrate nerve fibers or cross the blood–brain barrier, it is administered in an attempt to bind any circulating toxin outside the CSF.

(1) Doses range from 50,000 IU to 300,000 IU given intramuscularly or intravenously every 12 hours (it must be given within 10 hours of clinical signs).

(a) In adult horses, 50 ml of CSF is withdrawn from the atlanto-occipital (AO) site, and a similar volume of TAT (1000 IU/ml) is injected.

(b) In a foal, 30 ml is used. Also, 150 mg of methyl prednisolone succinate may be administered intrathecally, along with the TAT, to decrease irritation.

(2) The use of intrathecal TAT is controversial. Though some authors report success with intrathecal treatment, others report convulsions and death.

b. Cleansing the wound. Another component of treatment is to prevent further absorption of newly-formed toxin by thorough debridement and cleansing of the wound to allow oxygenation, which ensures a hostile environment for clostridial growth.

c. Sedatives. Uncontrolled muscle spasms and rigidity can be controlled through the use of sedatives (e.g., acetylpromazine, detomidine) or muscle relaxants (e.g., glyceryl guaiacolate).

d. Supportive care involves a heavily bedded, dark stall, minimal manipulation, fluid and electrolyte balance, and bowel and bladder evacuation. Cotton plugs in the ears decrease noise stimuli. A tracheostomy tube should be available for placement if laryngospasm occurs.

e. Penicillin. C. tetani is sensitive to penicillin. High doses given intravenously may help.

7. Prognosis. The response to treatment in horses and small ruminants is poor but cattle frequently recover. Recovery from clinical tetanus does not confer immunity.

8. Prevention

a Vaccination. A good vaccination program successfully prevents the disease.

b. Boosters with toxoid and antitoxin should be given at times of penetrating wounds.

c. Proper care and hygiene for tail docking and castration instruments is necessary.

B. Botulism

1. Patient profile. Adult cattle, sheep, and horses are periodically affected by botulism, whereas swine appear to be quite resistant. Outbreaks may occur in cattle that are given new batches of oats, rye, or corn silage, or the big grass bale silage. Foals 1–3 months old can also have a form of the disease called shaker foal syndrome.

2. Clinical findings

a In peracute cases, there is sudden death with no premonitory signs.

b. In acute cases, there is an abrupt onset of flaccid paralysis or tetraparesis that has no association with systemic illness or trauma.

(1) A progressive muscular weakness and paralysis may begin with weakness and ataxia and progress to the inability to rise. Muscle tremors and fasciculation associated with the weakness may be marked. Flaccid paralysis continues until the animal dies of respiratory arrest.

(2) Consciousness and sensation are retained until death. Affected animals appear to have normal sensation but depressed reflexes.

(3) Urine retention, ileus with constipation, and tachycardia are reported in cattle.

(4) Head signs can include pupillary dilation, tongue paralysis, and dysphagia. Dyspnea and cyanosis may also be noted, which are likely the result of impaired respiratory muscle function.

c. Foals with shaker foal syndrome are vigorous and rapidly growing, usually between 1 and 3 months of age.

(1) The main complaint is that they spend time lying down and walk with a stilted gait. As the disease progresses, the animal can only stand a few minutes at a time before trembling and falling.

(2) These foals have a slab-sided appearance because of the paralysis of the intercostal muscles.

(3) Terminally, inhalation pneumonia may occur.

3. Etiology and pathogenesis

a. Etiology. Botulism is caused by the effects of one of eight serologically distinct neurotoxins produced by *Clostridium* botulinum. Two syndromes are recognized in botulism toxicity: forage poisoning and toxicoinfectious botulism in foals.

(1) In forage poisoning, affected animals have ingested the preformed toxin in spoiled feed (with a pH greater than 4.5) or feed that has been contaminated with carrion. Dried poultry waste, sometimes used as a protein source or bedding for cattle, is another source of the toxin. Outbreaks also happen with new batches of oat, rye, and corn silage and by bale grass silage (anaerobic environment).

(2) Toxicoinfectious botulism of foals is caused by the colonization of C. botulinum type B in the gastrointestinal tract with subsequent toxin production within the intestine.

b. Pathogenesis. The neurotoxin causes muscle paralysis by blocking exocytosis of acetylcholine at the presynaptic membrane of the neuromuscular junction. Thus, only skeletal muscle is affected, resulting in flaccid paralysis. When the toxin has bound to the neuromuscular junction membrane, it must be metabolized before function can be regained.

4. Diagnostic plan

a. The clinical signs of acute onset of flaccid paralysis in the absence of other disease or trauma is highly suggestive of botulism. Decreased tongue tone may be prominent.

b. Because the toxin is so potent there is usually too little circulating toxin in large animals to be detected by laboratory methods. Samples of the feed ingested should be obtained and submitted to specialized laboratories for detection of the preformed botulinum toxin, where extraction and mouse inoculation can be performed.

5. Therapeutic plan

a. Supportive care. There is no effective antidote when the toxin has bound to the neuromuscular junction. Therefore, mildly affected animals require supportive care to allow time for toxin metabolism.

(1) Nutritional and fluid support can be administered via nasogastric tube, and urinary catheterization may be required.

(2) Recumbent animals. If the animal is recumbent, it should be kept in deep bedding and rolled frequently to prevent muscle ischemia.

b. Therapeutic agents

(1) Mineral oil and sodium sulfate. Any toxin that may be in the gastrointestinal tract can be eliminated more quickly by administering mineral oil or sodium sulfate.

(2) Activated charcoal may also be given to absorb the toxin within the gastrointestinal tract.

(3) Antitoxin administration may be beneficial to neutralize the circulating toxin that has yet to reach the neuromuscular junction, but these products are extremely expensive.

6. Prognosis. The prognosis for recovery when signs are observed is poor.

7. Prevention

a Vaccination against C. botulinum type B appears to be effective in areas where foals are affected (e.g., Kentucky).

b. Removal of the source. In forage poisoning, the source of the toxin must be found and eliminated because there is no available vaccine.

C. Equine motor neuron. disease

1. Patient profile. This recently-described disease affects adult horses between ages 2 and 25 years. This disease may be overrepresented in Quarter hones and is more commonly described in the northeastern United States.

2. Clinical findings include a marked weight loss, despite a ravenous appetite. This sign is preceded by an acute onset of fine muscle fasciculations, coarse trembling of limb muscles, and a tendency to lie down.

3. Etiology and pathogenesis
   a Etiology. Neither toxic nor infectious causes are yet ruled out.
   b. Pathogenesis. The clinical signs occur because of neurogenic muscle atrophy, in which there is weakness but no loss of proprioception. There is a widespread degeneration of somatic motor neurons in the ventral horn of the spinal cord. The changes noted are similar to amyotrophic lateral sclerosis (ALS) in humans.

4. Diagnostic plan and **differential** diagnoses. There is no definitive **antemortem** test; thus, it is important to rule out more treatable diseases, such as **rhabdomyolysis,** white muscle disease, and botulism.

5. Therapeutic plan and **prevention.** Because little is known of the pathogenesis of this disease, there are no guidelines for successful treatment or prevention.

D. Periodic spasticity of **cattle** (barn **cramps)**

1. Patient profile. This disease affects mature cattle (3–7 years old) of both sexes and all breeds, especially Holstein-friesian and Guernsey bulls.

2. Clinical findings
   a. Initial findings. In affected cattle, there are intermittent spastic contractions of the muscles of the back, neck, and legs. Attacks may last for several minutes.
   b. Progression of the disease may be accompanied by weight loss, increased recumbency, and prolonged spastic periods.

3. Etiology and pathogenesis. The etiology is unknown, but the disease is thought to be inherited as a single autosomal-dominant gene of incomplete penetrance. There is no significant neuromuscular pathologic change.

4. Diagnostic plan. Clinical signs are usually sufficiently characteristic to determine the diagnosis.

5. **Differential** diagnoses. Periodic spasticity must be differentiated from spastic paresis, trauma to the CNS, hypomagnesemia, and lameness problems.

6. Therapeutic plan. No effective treatment is available, but mephenesin may control severe signs for some weeks.

7. Prognosis. Complete recovery never occurs, and the signs are usually progressive.

8. Prevention. Because there may be a genetic association to this disease, there can be selection pressure placed against the occurrence of the disease, but this is unlikely to occur.

E. **Stringhalt, lathyrism,** and shivering

1. Patient profile. Shivering is a condition described most often in draft horses, whereas any breed of adult horse can be affected by stringhalt or lathyrism.

2. Clinical **findings**
   a. In stringhalt, there is an abrupt onset of excessive flexion of the hindlegs that may progressively worsen. This can occur as an outbreak, particularly when related to the ingestion of sweet pea plants **(lathyrism).**
   b. In shivering, there are mild muscle tremors to the hind quarters and tail that

occur with movement. Signs are exaggerated when the horse is asked to back up. Some horses are unable or unwilling to back up. It may be impossible to lift the hind legs.

3. Etiology and pathogenesis. Many causes have been proposed for these problems, but the underlying feature is likely some alteration of the neuromuscular spindle and gamma efferent fibers to alter the control of muscle tone.

4. Diagnostic plan and differential diagnoses. Diagnosis is based on clinical signs, which must be differentiated from upward fixation of the patella.

5. Therapeutic plan. There is no treatment, and even muscle relaxants have not been useful in relieving the signs. Horses with stringhalt should be removed from access to any toxic plants.

6. Prognosis. The prognosis is unfavorable for shivering, but many cases of horses with sporadic stringhalt slowly improve with time.

V. **LOCALIZED** PERIPHERAL NERVE DISORDERS

Spastic paresis **(Elso** heel, bovine spastic paralysis)

1. Patient profile. This disorder occurs in calves at 6 weeks to 8 months of age. The Holstein breed is more frequently affected, but spastic paresis can be seen in other breeds.

2. Clinical findings
   a Gait change. Affected calves walk with straight hind limbs with the angle of the hock close to 180°, but there is no pain associated with this gait change. Usually one hind limb is more severely affected, causing it to be held extended behind the calf at rest, swinging like a pendulum, and bearing little weight.
   b. As the disease progresses, there is gluteal atrophy and a raised tail head. The gastrocnemius and Achilles tendon are tense. Involuntary leg jerking may occur, and there is an arched back, flexion of the carpi flex, and weight loss.

3. Etiology. The cause is unknown, but certain famous sires (e.g., Elso II, hence the name Elso heel) have been suggested as genetic carriers of this problem. Environmental factors may also contribute.

4. Diagnostic plan. Diagnosis is made based on clinical signs and the absence of pain.

5. Differential diagnoses. Spastic paresis must be differentiated from gonitis, dorsal luxation of the patella, tarsitis, and fractures.

6. Therapeutic plan. Partial or total tibial neurectomy is the treatment of choice and is esthetically pleasing.

7. Prognosis. The prognosis for recovery is poor without some intervention.

B. ***Sorghum*** cystitis and ataxia

1. Patient profile and history. This disorder can occur in any age or breed of horse with a history of ingesting sorghum ensilage, fodder, or grain.

2. Clinical findings. The main clinical sign is cystitis with urinary incontinence and urine scalding. There may also be ataxia, which becomes exaggerated when the animal is backed up or turned.

3. Etiology and pathogenesis. Ingestion of the growing Sorghum plant may result in mortality due to the high cyanide or nitrate poisoning. The cause of the cystitis and ataxia is unknown, but white-matter degeneration was observed in the spinal cord.

4. Diagnostic plan. Diagnosis is based on the clinical signs of cystitis and urinary incontinence, accompanied by access to Sorghum.

5. Therapeutic plan. There is no effective treatment.

6. Prognosis. When signs are present, the prognosis is grave.

C. **Faciovestibular** nerve disease in horses

1. Patient profile and history. All reported cases of this disease have been in mature horses (older than 4 years). Many cases have involved aged horses, and there is usually no history of trauma.

2. Clinical findings. There is an acute onset of unilateral paralysis of the facial and vestibular nerves.
   a. The facial nerve signs include an ear droop, ptosis, muzzle deviation, or all of these signs. Exposure keratitis of the affected eye may occur.
   b. Vestibular nerve signs include head, body tilt, or both; reluctance to move; and ataxia of the limbs (with limbs on the ipsilateral side of the lesion more flexed than the contralateral limbs). Disturbances of balance due to the vestibular changes can be accentuated by blindfolding the animal.
   c. General signs. Affected horses are very anxious, and the signs may progress to an inability to stand.

3. Pathogenesis. The neurologic signs are attributed to an acute pathologic fracture of the petrous temporal bone. These signs are secondary to a chronic bony proliferation, and fusion is the result of a chronic otitis media. When fusion has occurred, a transfer of forces from tongue movement may be enough to cause a fracture of the petrous temporal bone, which may extend into the cranial vault near the internal acoustic meatus. Damage of cranial nerves VII and VIII occurs here.

4. Diagnostic plan and laboratory tests
   a. Skull radiographs reveal a thickening of the shaft of the stylohyoid bone, extending to the temporohyoid articulation.
   b. Endoscopy of the guttural pouches may also illustrate a thickened stylohyoid bone.

5. Differential diagnoses include central vestibular disease caused by abscess or tumor, EPM, trauma, and otitis.

6. Therapeutic plan. Prophylactic antibiotic therapy has been suggested but is only of limited success.

7. Prognosis. The prognosis for recovery is guarded, but affected horses can compensate visually for vestibular deficits over time.

D. Radial nerve **paralysis**

1. Patient profile. All species of any age can incur this nerve damage.

2. Clinical findings
   a. In partial paralysis, there is an inability to advance the limb, but if the foot is placed, then the animal can support its weight.
   b. In total paralysis, there is a "dropped" elbow and an inability to advance the limb. The animal cannot bear weight, and skin sensation over the dorsal and medial aspects of the metacarpus and phalanges is reduced.

3. Etiology. Injury to the nerve frequently occurs when heavy animals are restrained in lateral recumbency without adequate padding. The nerve injury may also be caused by a fractured humerus or a severe blow to the lateral aspect of humerus.

4. Diagnostic plan. The neurologic examination reveals changes of radial nerve deficits.

5. Therapeutic plan. Rest and time may be beneficial for recovery. Following acute radial nerve injury, it is advisable to administer some form of anti-inflammatory agent to decrease local tissue swelling around this nerve.

E. **Suprascapular** paralysis **(sweeny)**

1. Patient profile. Draft horses that pull in harnesses or cows that are crowded through a narrow opening can readily damage the suprascapular nerve.

2. Clinical findings. The most prominent changes noted include atrophy of supraspinatus and infraspinatus muscles. The stride may be shortened, but weight bearing is unaffected.

3. Etiology. This injury was seen in draft horses that wore heavy collars. Crowding through a doorway may result in trauma to this area.

4. Diagnostic plan. The neurologic examination, which reveals selected muscle atrophy, is usually sufficient for diagnosis.

5. Therapeutic plan. Apart from the use of anti-inflammatory drugs for trauma, surgical creation of a notch in the scapula has been a suggested treatment, as has the removal of any entrapped tissue.

6. Prognosis. The prognosis is guarded, but some acutely affected animals spontaneously recover.

F. **Obturator-sciatic paralysis**

1. Patient profile. This is a common calving injury that is seen most often in heifers.

2. Clinical findings. Cows and heifers exhibit various degrees of hind limb ataxia, with wide displacement of the hind limbs and frequent kicking of the hindleg. The paralysis is usually bilateral, with uncontrolled abduction of the hind limbs and recumbency, followed by an inability to rise. Provided no other injury is present, there appears to be normal sensation to the hind limbs.

3. Etiology and pathogenesis. This paralysis occurs predominately in cattle at parturition as a result of calving trauma. Both the obturator and the L6 branch of the sciatic nerve innervate the adductor muscles of the hind limbs, incurring trauma during calving.

4. Therapeutic plan. Tying the hind limbs approximately 1 meter apart prevents excessive abduction and often enables an animal to stand. Muscle massage and rolling the recumbent animal helps decrease decubital ulcer formation.

G. Cauda equina neuritis **(polyneuritis equi)**

1. Patient profile and history
   a. Patient profile. This relatively uncommon problem occurs in horses and is reported as urinary and fecal incontinence, possibly with colic or rubbing of the tail head. The disease affects adult horses, with no breed or sex predilection. There is a wide age range, although it has not been reported in foals or aged horses.
   b. History. A recent vaccination or respiratory illness has been a frequently-mentioned aspect of reported cases.

2. Clinical findings
   a. The initial findings on physical examination are urine scald to the perineum, obstipation, and broken tail head hairs. On inspection of the tail region, there is a ring of hypersensitivity surrounding an area of analgesia. A loss of tail tone with paralysis of the bladder, rectum, anal sphincter, and penis or vulva is evident.
   b. A head tilt and facial paralysis are possible but uncommon.
   c. Pelvic limb gait abnormalities, if present, are subtle.

3. Pathogenesis. This granulomatous inflammatory disease occurs at the level of the extradural nerve roots and may be an autoimmune disease. Some horses have been found to have circulating antibodies against P_2-myelin protein. Postmortem examination reveals a thickened and fibrotic cauda equina. A similar but less severe reaction may occur at the cranial nerve level.

4. Diagnostic plan and laboratory tests
   a. Clinical signs of localized hyperesthesia with analgesia around the tail region are typical for cauda equina neuritis.

b. The **CSF** sample may be normal or may have some protein and cellular increases, but this test cannot confirm the disease.

c. Biopsy. A caudal epidural biopsy is required for antemortem diagnosis.

5. Differential diagnoses. A thorough rectal examination should be done to rule out a fractured sacrum, the most important differential diagnosis for such clinical signs.

6. Therapeutic plan and prognosis

a. Supportive **care.** With supportive care of regular bowel and urinary bladder evacuation, affected horses have been maintained for up to 1 year.

b. Euthanasia. The signs progress, despite glucocorticoid treatment, and necessitate euthanasia.

STUDY QUESTIONS

DIRECTIONS: Each of the numbered items or incomplete statements in this section is followed by answers or by completions of the statement. Select the ONE numbered answer or completion that is BEST in each case.

1. Which one of the following statements regarding listeriosis is true?

(1) It is known as a circling disease when it affects cattle and horses.

(2) Heavy silage feeding is a particular risk factor because the cool acid conditions (i.e., a pH less than 4.5) favor growth.

(3) The changes in the brain are usually multifocal microabscesses, which can cause several cranial nerve deficits as well as depression.

(4) The organism gains entrance to the nervous tissue by intestinal penetration and embolization to cause multifocal brain abscesses.

(5) When signs are evident in cattle, the animals are unlikely to respond to treatment.

2. Regarding pseudorabies or rabies in large animals, which one of the following statements is true?

(1) All farm animals can be affected by both pseudorabies and rabies.

(2) Pigs can carry the pseudorabies virus, with adults showing mild or subclinical signs.

(3) Similar to rabies, pseudorabies is spread primarily by direct bite wounds from infected animals.

(4) Both pseudorabies and rabies are sporadic rare diseases across North America.

(5) Both pseudorabies and rabies are caused by antigenically similar rhabdoviruses.

3. Which one of the following statements regarding some central nervous system (CNS) diseases of the horse is correct?

(1) In arboviral encephalitis, such as eastern or western equine encephalitis (EEE, WEE), the main source of spread is mosquito that have ingested the viruses from clinically ill horses.

(2) Yellow star thistle can cause rigidity of muscles of mastication in the horse, but signs in cattle are photosensitization.

(3) Equine encephalitis viruses can also affect people; thus, an infected horse should be treated by isolation.

(4) Equine viral encephalitis occurs mainly in summer months in northern climates.

(5) Russian knapweed toxicity in horses can result in starvation because of the induction of paralysis of pharyngeal muscles.

4. Which statement regarding the slow virus diseases in large animals [i.e., scrapie and bovine spongiform encephalopathy (BSE)] is correct?

(1) Both diseases have a long incubation period, with the spread mainly via contaminated animal feeds.

(2) Neither disease occurs in the animal population of North America.

(3) Of the two diseases, BSE is currently of more economic and public health significance.

(4) Both diseases cause a characteristic intense pruritus and emaciation as key clinical abnormalities.

5. Regarding tetanus and botulism in large animals, which one of the following statements is correct?

(1) Horses are highly susceptible to both toxins, with clinical disease occurring in cattle, sheep, and pigs being progressively more common.
(2) For both botulism and tetanus, the administration of specific antitoxin serum can neutralize the toxin at the site of binding.
(3) A horse that recovers from clinical tetanus is subsequently immune to this disease.
(4) Diagnosis of botulism in a cow is usually made by the demonstration of botulinum toxin in the serum.
(5) Botulism in large animals has been associated with feeding newly made silage or big round bale silage, whereas pasture-induced hypomagnesemia could be mistaken for tetanus.

.6. When comparing swayback (enzootic ataxia) to caprine arthritis encephalitis (CAE), which one of the following statements is correct?

(1) Swayback occurs in newborn lambs, whereas paresis from CAE is seen only in kid goats.
(2) Swayback exhibits lower motor neuron (LMN) signs with hyporeflexia and depressed reflexes, whereas CAE is mainly seen with upper motor neuron (UMN) signs.
(3) CAE has no cure, but, because swayback is caused by copper deficiency, replacement of copper stores allows affected lambs to return to normal.
(4) Both swayback and CAE are mainly associated with intrauterine processes or infection.

7. Which one of the following statements regarding equine protozoal myeloencephalitis (EPM) is correct?

(1) The causative organism has been identified as *Sarcocystis falcatula,* a parasite whose definitive host is the opossum and whose intermediate hosts are birds.
(2) The parasite only encysts in the central nervous system (CNS) of horses by accident; thus, this parasite only poses a risk if the nervous tissue is eaten by the definitive host.
(3) The presence of antibody to the parasite in the cerebrospinal fluid (CSF) is definitive proof of CNS infection in a seropositive horse.
(4) The drugs of choice are amprolium or spiramycin (another coccidiostat).
(5) Experimentally, the signs of disease first occur when the parasite is undergoing destruction by the immune system within the CNS.

8. Which set of neurologic diseases in large animals warrants isolation of diseased animals to prevent the spread to others in the herd or flock?

(1) Bovine spongiform encephalopathy (BSE), rabies, equine protozoal myeloencephalitis (EPM)
(2) Pseudorabies, listeriosis, nervous coccidiosis, eastern equine encephalitis (EEO
(3) Caprine arthritis encephalitis (CAD, scrapie, equine herpesvirus-1, EPM
(4) Equine degenerative myelopathy (EDM), thromboembolic meningoencephalitis (TEME), CAE
(5) Pseudorabies, CAE, rabies

9. Which set of neurologic signs and their corresponding etiologies is correct?

(1) In horses that have ingested yellow star thistle or Russian knapweed, there may be an acute onset of flaccidity of the muscles of mastication, resulting in a characteristic grimacing expression of the facial muscles with drooping lips and nostrils.
(2) For leukoencephalomalacia in horses, corn being fed with large amounts of *Fusarium* species is a strong suggestion of the diagnosis in up to 80% of cases.
(3) In hepatic encephalopathy of large animals, affected animals are most commonly neonates because of portosytemic shunts, with signs of depression, head pressing, and yawning that may wax and wane depending on the amount of time since eating.
(4) With vitamin A deficiency, there can be peripheral blindness in calves, with absent pupillary light reflexes and dilated pupils in severe cases. These signs are likely the result of the distortion of bone development involving the optic nerve.
(5) In cauda equina neuritis, initial physical examination findings include urine scald, bladder and tail paralysis, and a central area of hypersensitivity surrounded by a ring of analgesia in the tail region.

10. Which one of the following statements regarding the clostridial diseases in large animals is correct?

(1) Botulism is caused by the effects of one of eight serologically distinct neurotoxins that causes muscle paralysis by blocking acetylcholine at the presynaptic membrane of the neuromuscular junction. Thus, botulism affects only skeletal muscle.
(2) In botulism caused by forage poisoning, affected animals have ingested the preformed toxin in spoiled feed (with a pH less than 4.5) or feed such as new batches of oat, rye, and corn silage or bale grass silage that has had oxygen damage.
(3) The diagnosis of botulism requires the demonstration of a circulating toxin in large animals, and samples of the feed ingested seldom have the toxin identified.
(4) Tetanus is diagnosed based on the clinical signs of hyperesthesia to external stimuli, a demonstrating circulating neurotoxin, and the isolation of the organism from contaminated wounds.
(5) *Clostridium tetani* produces a neurotoxin that migrates along nerve fibers and in blood and lymph fluid from its site of production and acts mainly at the neuromuscular junction to cause muscular spasms.

ANSWERS AND EXPLANATIONS

1. The answer is 3 [II D 3 a-c]. The changes in the brain are usually multifocal microabscesses. Horses are not commonly affected. Decaying organic matter and spoiled silage with a pH more than 5.0 favor growth. The causative organisms enter via abraded buccal mucosa. The prognosis with antibiotic therapy is fair to good in cattle.

2. The answer is 2 [II D 5 a-b; 6 a]. Adult pigs show mild signs when carrying the pseudorabies virus. Not all animals can be affected by both diseases; hones are the exception for pseudorabies. Pseudorabies is spread by contact with abraded skin or through the respiratory tract. Pseudorabies is exotic to Canada, and both rabies and pseudorabies can be 'more prevalent in certain geographic locales. Pseudorabies is a herpes, which thus can become latent

3. The answer is 4 [II D 7; 8 b]. Because equine encephalitis is spread by insects, disease occurs in the summer months in northern climates. The reservoirs for arboviral encephalitis are birds, not mosquitos. Yellow star thistle can cause rigidity of muscles of mastication in the horse, but cattle are unaffected. Infected horses do not pose a risk, though people can get this infection. Russian knapweed toxicity can cause horses to starve because of rigid muscles of mastication, not paralysis.

4. The answer is 3 [II E 1 c; 2 a]. Bovine spongiform encephalopathy (BSE) is of more significance because of the large-scale slaughter of mature cattle and the possible link to human dementia. Scrapie is thought to be contracted mainly from vertical or horizontal spread. Scrapie occurs sporadically and is found in sheep flocks. Pruritus is only prominent in scrapie.

5. The answer is 5 [IV A 1; B 1, 3]. Cattle are the least susceptible to tetanus but are highly susceptible to botulism. When bound, the toxin must be metabolized. There is insufficient toxin from clinical tetanus to induce immunity. The amount of circulating botulinum toxin is usually insufficient for detection.

6. The answer is 2 [III B 1 c; 2 a-c]. Swayback also occurs in kids and piglets. The neurologic damage in swayback is most likely permanent and will not be cured by the replacement of copper. CAE is mainly Spread by postnatal contact through colostrum or milk.

7. The answer is 1 [III A 3 c]. The causative organism has been identified as *Sarcocystis falcatula.* There appears to be no encysting; thus, there is no Infective stage from the horse. Serum contamination of the cerebrospinal fluid sample could give false-positive results. The drugs of choice are pyrimethamine and sulfas. The parasite causes cell neuronal and microglial damage independently, and the immune reaction in the CNS is not vigorous or rapid.

8. The answer is 5 [II D 5, 6 c; III B 1 c]. Pseudorabies, caprine arthritis encephalitis (CAE), and rabies can be spread from infected animals. Bovine spongiform encephalopathy (BSE) is spread in feedstuffs and equine protozoal myelitis (EPM) is not spread from horses. Nervous coccidiosis is sporadic and is only associated with coccidiosis, and equine encephalitis virus is a "dead-end host" in the horse. CAE, scrapie, and equine herpesvirus-1 can theoretically be spread through contact, but EPM is not spread from horses. Equine degenerative myelopathy (EDM) is a noninfectious disease related to vitamin E problems, and thromboembolic meningoencephalitis (TEME) is not thought to be infectious from an infected animal (although it can be carried in other animals).

9. The answer is 4 [II D 10 b-c]. With vitamin A deficiency, there can be peripheral blindness in calves, with absent pupillary light reflexes and dilated pupils in severe cases. Signs include rigid muscles and lips that are drawn back, not drooping features. This mold *(Fusarium)* can be found in up to 80% of samples. Hepatic encephalopathy is uncommon in neonates and is usually seen in adult animals. There is a central area of analgesia surrounded by a ring of hypersensitivity.

10. The answer is 1 [VIII B 3, 41. Botulism affects only skeletal muscle. Feed is spoiled above pH 4.5, and the toxin needs an anaerobic environment. There is too little circulating toxin for detection, so feed samples are preferred for testing. culture from the wound is difficult, and it is not readily possible to demonstrate a toxin in circulation because most toxins are bound. The main site of action is at the inhibitory interneurons of the spinal cord. The inhibitory interneurons predominate involve the antigravity (or extensor) muscles, and, thus, the signs of tetanus reflect the overactivity of these muscles.